Exhibit 28

IN THE UNITED STATES DISTRICT COURT
FOR THE SOUTHERN DISTRICT OF NEW YORK

- - -

IN RE: ACETAMINOPHEN - ASD-ADHD PRODUCTS LIABILITY LITIGATION	:	MDL NO. 3043
	:	CASE NO. 1:22-md-03043 -DLC
THIS DOCUMENT RELATES TO: ALL CASES:	:	Judge Denise Cote
1:22-md-03043	:	

- CONFIDENTIAL -
- PURSUANT TO PROTECTIVE ORDER -

- - -

September 1, 2023

- - -

Videotaped deposition of ALEX KOLEVZON, M.D., taken pursuant to notice, was held at the Best Western Plus & Venue, 503 Washington Avenue, Kingston, New York, beginning at 8:31 a.m., on the above date, before Michelle L. Gray, a Registered Professional Reporter, Certified Court Reporter, Certified Realtime Reporter, and Notary Public.

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GOLKOW LITIGATION SERVICES
877.370.3377 ph| 917.591.5672
deps@golkow.com

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APPEARANCES:

WATTS GUERRA LLP
BY: MIKAL C. WATTS, ESQ.
(In person)
BY: HAILEY WATTS, ESQ.
(In person)
BY: SHELLY SANFORD, ESQ.
(Zoom)
BY: JOHN CRACKIN, ESQ.
(Zoom)
BY: JERRY WHITE, ESQ.
(Zoom)
875 East Ashby Place
Suite 1200
San Antonio, Texas 78257
(210) 447-0300
mcwatts@wattsguerra.com
hwatts@wattsguerra.com
ssanford@wattsguerra.com
jwhite@wattsguerra.com
Representing the Plaintiffs

BEASLEY ALLEN LAW FIRM
BY: W. ROGER SMITH, III, ESQ.
(In person)
218 Commerce Street
Montgomery, Alabama 36104
(334) 954-7555
roger.smith@beasleyallen.com
Representing the Plaintiffs

THE LANIER LAW FIRM
BY: CATHERINE HEACOX, ESQ.
(Zoom)
BY: CRISTINA DELISE, ESQ.
(Zoom)
10940 West Sam Houston Parkway North
Suite 100
Houston, Texas 77064
(713) 659-5200
catherine.heacox@lanierlawfirm.com
cristina.delise@lanierlawfirm.com
Representing the Plaintiffs

Page 3

APPEARANCES: (Cont'd.)

THE LANIER FIRM
BY: EVAN M. JANUSH, ESQ.
(Zoom)
126 East 56th Street
6th Floor
New York, New York 10022
(212) 421-2800
evan.janush@lanierlawfirm.com
Representing the Plaintiffs

KRAUSE & KINSMAN LAW FIRM
BY: TRICIA CAMPBELL, ESQ.
(Zoom)
4747 Grand Avenue
Suite 300
Kansas City, Missouri 64112
(816) 200-2900
tcampbell@krauseandkinsman.com
Representing the Plaintiffs

TRACEY FOX & WALTERS
BY: SEAN P. TRACEY, ESQ.
(Zoom)
BY: LAWRENCE TRACEY, ESQ.
(Zoom)
440 Louisiana Street
Unit 1901
Houston, Texas 77002
(713) 489-6304
stracey@traceylawfirm.com
ltracey@traceylawfirm.com
Representing the Plaintiffs

Page 4

APPEARANCES: (Cont'd.)

KELLER POSTMAN LLC
BY: ASHLEY BARRIERE, ESQ.
(Zoom)
BY: AMANDA HUNT, ESQ.
(Zoom)
BY: ROSEANN ROMANO, ESQ.
(Zoom)
BY: J.J. SNIDOW, ESQ.
(Zoom)
BY: REBECCA KING, ESQ.
(Zoom)
150 North Riverside Plaza
Suite 4100
Chicago, Illinois 60606
(312) 741-5220
ashley.barriere@kellerpostman.com
amanda.hunt@kellerpostman.com
roseann.romano@kellerpostman.com
JJ.snidow@kellerpostman.com
Rebecca.king@kellerpostman.com
Representing the Plaintiffs

HOLWELL, SHUSTER & GOLDBERG, LLP
BY: EILEEN MONAGHAN DELUCIA, ESQ.
(Zoom)
BY: DANIEL SULLIVAN, ESQ.
(Zoom)
425 Lexington Avenue
New York, New York 10017
(646) 837-5151
edelucia@hsgllp.com
Dsullivan@hsgllp.com
Representing the Plaintiffs

Page 5

APPEARANCES: (Cont'd.)

HOLWELL, SHUSTER & GOLDBERG, LLP
BY: EILEEN MONAGHAN DELUCIA, ESQ.
(Zoom)
BY: DANIEL SULLIVAN, ESQ.
(Zoom)
425 Lexington Avenue
New York, New York 10017
(646) 837-5151
edelucia@hsgllp.com
Dsullivan@hsgllp.com
Representing the Plaintiffs

SKADDEN, ARPS, SLATE, MEAGHER & FLOM LLP
BY: ALLISON M. BROWN, ESQ.
(In person)
BY: JOSEPH A. CARUSO, ESQ.
(In person)
One Manhattan West
New York, New York 10001
(212) 735-3000
allison.brown@skadden.com
joseph.caruso@skadden.com
Representing Johnson & Johnson Consumer Inc. (JJCI)

BUTLER SNOW LLP
BY: DAVID M. COHEN, ESQ.
(Zoom)
BY: RAQUEL LUCAS, ESQ.
(Zoom)
810 Seventh Avenue
Suite 1105
New York, New York 10019
(646) 606-2996
david.cohen@butlersnow.com
raquel.lucas@butlersnow.com
Representing Johnson & Johnson Consumer Inc. (JJCI)

APPEARANCES: (Cont'd.)

KING & SPALDING LLP
BY: LUKE BOSSO, ESQ. (Zoom)
1700 Pennsylvania Avenue, NW
Suite 900
Washington, D.C. 20006
(202) 737-0500
lbosso@kslaw.com
Representing the Defendant, Walmart, Inc., and Wal-Mart Stores, Inc.

BARNES & THORNBURG LLP
BY: NADINE S. KOHANE, ESQ. (Zoom)
390 Madison Avenue
12th Floor
New York, New York 10017
(646) 746-2000
nkohane@btlaw.com
Representing CVS Pharmacy, Inc., CVS Health Corporation, Walgreen Co., Walgreens Co., and Walgreens Boots Alliance, Inc.

BARNES & THORNBURG LLP
BY: SANDRA KO, ESQ. (Zoom)
BY: DEANNA LEE, ESQ. (Zoom)
555 12th Street N.W.
Suite 1200
Washington, D.C. 20004
(202) 289-1313
sko@btlaw.com
deanna.lee@btlaw.com
Representing the Defendant, Costco Wholesale Corporation

APPEARANCES: (Cont'd.)

STONE DEAN LLP
BY: JOSEPH A. LARA, ESQ. (Zoom)
21052 Oxnard Street
Woodland Hills, California 91367
(818) 999-2232
jlara@stonedeanlaw.com
Representing the Defendant, The Kroger Co.

MORRISON & FOERSTER LLP
BY: LINDSEY HAAS CAIN, ESQ. (Zoom)
4200 Republic Plaza
370 Seventeenth Street
Denver, Colorado 80202
(303) 592-1500
lcain@mofo.com
Representing the Defendant, Target Corporation

HAIGHT BROWN & BONESTEEL LLP
BY: KATIE TRINH, ESQ. (Zoom)
555 South Flower Street, 45th Floor
Los Angeles, California 90071
213.542.8000
ktrinh@hbblaw.com
Representing the Defendant, Big Lots Stores-PNS, LLC

DUANE MORRIS, LLP
BY: SEAN K. BURKE, ESQ. (Zoom)
901 New York Avenue N.W.
Suite 700 East
Washington, DC 2000
202.776.5236
Sburke@duanemorris.com
Representing the Defendant, Dollar General Corporation

APPEARANCES: (Cont'd.)

ALSO PRESENT:
VIDEOTAPE TECHNICIANS:
Henry Marte - Golkow (In person)
Danny Ortega - Golkow (In person)
LITIGATION TECHNICIAN:
Erik Thorsnes - U.S. Legal (In person)
Jason Short - IT (Zoom)

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I N D E X

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Testimony of:

ALEX KOLEVZON, M.D.

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E X H I B I T S

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DEPOSITION SUPPORT INDEX

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THE VIDEOGRAPHER: We are now on the record.

My name is Danny Ortega. I'm the legal videographer for Golkow Litigation Services.

Today's date is September 1st, 2023, and the time is 8:31 a.m.

This video deposition is being held at 503 Washington Avenue, Kingston, New York, in the matter of Tylenol, Acetaminophen/Tylenol ASD-ADHD products liability litigation MDL.

The deponent today is Alex Kolevzon.

All counsel will be noted on the stenographic record.

The court reporter today is Michelle Gray and will now swear in the witness.

- - -

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... ALEX KOLEVZON, M.D., having been first duly sworn, was examined and testified as follows:

- - -

EXAMINATION

- - -

BY MR. WATTS:

Q. What is your name?

A. Alex Kolevzon.

Q. You are a medical doctor?

A. I am.

Q. Clinical psychiatrist?

A. I am.

Q. Okay. I'm here to take your deposition because you've been designated as an expert witness by the defendants in this case.

Do you understand that?

A. Yes.

(Document marked for identification as Exhibit Kolevzon 400.)

BY MR. WATTS:

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Q. Let me put up on the screen Exhibit 400. This is the amended notice for your deposition.

On Page 2 it shows the location at the Best Western Plus & Venue here in Kingston, New York; is that right?

A. Yes.

Q. And as I understand, we're in Kingston, New York, because we're near various parties' lake houses in here in the Catskills, right?

A. Yeah, minus the lake.

Q. Minus the lake? Okay.

A. No lake.

Q. But we're in what's known as the Catskills?

A. Right.

Q. Okay. Great.

Attached to that notice is what's known as a subpoena duces tecum. If we go to Page 7, it asks for documents to be produced.

Do you see that, sir?

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A. Yes.

Q. And without reading it all in, Number 1 asks for a copy of your CV, together with a list of papers you've written over the last ten years; is that right?

A. Yes.

Q. And if we go to Exhibit 401.

(Document marked for identification as Exhibit Kolevzon 401.)

BY MR. WATTS:

Q. This is the defendants' responses. And we can see on Page 3 of 17 that in response to that Request Number 1, the defendants refer us to your expert report and Rule 26 disclosures dated July 21 of 2023; is that right?

A. That's what the response says, yes.

Q. Okay. And I want to take you to that.

(Document marked for identification as Exhibit

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Kolevzon 405.)
BY MR. WATTS:
Q. Exhibit 405.
Is Exhibit 405, Exhibit 3 to your disclosure, if you go to the second page, your curriculum vitae that was provided to us in this case?
MS. BROWN: And, Mr. Watts, if we wanted to give the witness hard copies of what you're pulling up, the numbers correspond to the box?
MR. WATTS: They do.
MS. BROWN: Okay.
MR. WATTS: Everything is premarked. It's 400 through 568, and you're welcome to pull them out. I just figured I'd give you the use of them so we don't have to walk across the room every time.
MS. BROWN: Yep.
BY MR. WATTS:
Q. Is Exhibit 405 a true and

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correct copy of the curriculum vitae that was provided to us in this case, together with the Rule 26 disclosure, on July 21, 2023?
A. This is certainly a copy of my CV. I don't know whether it's the most up-to-date version.
Q. Well, let me ask you about that. If we go to Page 8 of the CV.
MS. BROWN: Here is the CV, 405. Is that what you have?
THE WITNESS: Yeah.
BY MR. WATTS:
Q. At the bottom, we see a list of publications that are numbered starting with 1 on Page 8; is that right?
A. Yes.
Q. And if we go all the way, just flip, Page 8, 9, 10, 11, 12, all the way to 18, we see your publications and the number growing one at a time until we get to Page 18.
A. Yes.
Q. And it goes through

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Publication Number 120, which is a paper with first author of Levy, and it's dated July 16th of 2023; is that right?
A. Yes.
Q. So that publication occurred about five days before your disclosure in this case on July the 21st; is that right?
A. Yes.
Q. Okay. And right under the publications there's a section dealing with books and chapters in books; is that right?
A. Yes.
Q. And if we continue from Page 18 to Page 19, Page 20, at the bottom of Page 19, the last book that's listed is one by Shapiro, Gibbs, and yourself, and it's published in 2022; is that right?
A. That's the reference. I think it's probably not available yet.
Q. Okay. Now I want to go to the Elmo for a second. And if you could,

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tell me whether or not the second edition of the Textbook of Autism Spectrum Disorders, by Hollander, Hagerman and Ferretti is listed under your books and book chapters.
A. I don't believe that it is.
Q. Okay. And just for references, going back to the presenter, Exhibit 542 is an e-mail to one of my partners by the publisher of this book.
(Document marked for identification as Exhibit Kolevzon 542.)
BY MR. WATTS:
Q. 542.
(Document marked for identification as Exhibit Kolevzon 494.)
MS. BROWN: We'll just give you a chance to get the hardcopy if you want it, Doctor.
MR. WATTS: I don't think he's going to need this one. It's real -- pretty short.

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BY MR. WATTS:
Q. Anyway, if this book, second edition of the Textbook of Autism Spectrum Disorders, it tells us was published on March 15th of 2022.
Do you see that, sir?
A. Yes.
Q. If we go to this book, you wrote a chapter in this book.
A. I didn't write a chapter. I was a co-author on a chapter.
Q. Co-author? And if we go to Chapter 11, we can see Chapter 11 is something entitled "Prenatal, Perinatal, and Parental Risk Factors."
And you are listed as a co-author of this book chapter of the second edition of the Textbook of Autism Spectrum Disorders that was published in March of 2022; is that right?
A. I'm listed as a co-author, yes.
Q. And in the chapter where you are a co-author, part of what is written

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is very "strong evidence that nonheritable prenatal or perinatal events are likely to have an etiological role," and it cites to Bristol in 1996; is that right?
MS. BROWN: Counsel, could you zoom in a little bit? It's a little hard to see on the screen.
MR. WATTS: I sure can.
MS. BROWN: Thank you.
MR. WATTS: Yeah.
MS. BROWN: Can you see it?
THE WITNESS: Yeah.
MS. BROWN: Okay.
THE WITNESS: I've read this chapter.
BY MR. WATTS:
Q. And part of what's written in the book chapter that you are a co-author of is "only half of the risk is explained by genetics"; is that right?
A. That's what was written by the person who wrote the article, the chapter.

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Q. You are listed as a co-author on that, right?
A. Yes. Although I didn't have the opportunity to review this before it was published.
Q. And if we can go to Page 187. On Page 187, this chapter where you are a co-author says, "We present plausible biological mechanisms linking those risk factors to ASD and suggest some directions for future research."
Did I read that correctly?
A. That's what the person who wrote the chapter wrote, yes.
Q. And that's what's in the chapter where you are listed as a co-author, right?
A. Yes. Although I don't agree with everything that was written in the chapter.
Q. And if we go to Page 191, the chapter where you are a co-author, published in March of 2022, has a section

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on acetaminophen, does it not?
A. The author of the chapter wrote a section on acetaminophen, yes.
Q. And the chapter where you are a co-author of this book chapter, published in March of 2022, says, "One analgesic and antipyretic medication classified in the B category for safety during pregnancy has recently been demonstrated to be associated with ASD and ADHD."
Do you see that?
A. That's what the person who wrote the chapter believes, yes.
Q. And it also says, "It has also been suggested that acetaminophen increases the risk for ASD by causing neuronal oxidative stress"; is that right?
A. Well, I don't believe that that's correct, but that person who wrote the chapter thinks it was correct.
Q. And you are listed as a co-author in the chapter where that

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statement exists, right, just last year?
A. I was listed as a co-author as a courtesy because I wrote the original chapter from the first edition of this textbook.
Q. Now if we go to Page 192.
MS. BROWN: What exhibit is this, Mr. Watts?
MR. WATTS: 494. It's the one that's in your box.
BY MR. WATTS:
Q. Page 192 says, "These findings suggest that metal toxicant update [sic] and essential element deficiency during specific developmental windows increases ASD risk and severity, supporting the hypothesis of systemic elemental dysregulation in ASD."
Is that what's said in here?
A. So the author of this chapter is talking about a hypothetical mechanism for increasing the risk, yes.
Q. But are the words that I read in the book chapter where you were

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listed as a co-author just last year?
A. You read the words correctly.
Q. Okay. Now, here is my question. When is the last time you testified as an expert in a lawsuit?
MS. BROWN: Objection to form.
THE WITNESS: I testified as an expert in a lawsuit in the late spring, early summer.
BY MR. WATTS:
Q. And that was in Beaumont, Texas?
A. No.
Q. Where was it?
A. It was --
Q. Galveston, Texas.
A. So you're talking about a different case --
Q. Okay.
A. -- which was in, I think, February of 2023. But that wasn't the last time I testified.

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Q. All right. Let's talk about the one in Texas.
A. Okay.
Q. That was a case where you were designated as an expert witness and there was an allegation of heavy metal ingestion being causative of autism spectrum disorder, right?
A. That was a case where the claim was that a child got autism because of eating baby food and that the heavy metals in the baby food caused him to have --
Q. And did you disclose that you were a co-author in this book chapter before you went to federal court and testified with respect to that subject?
MS. BROWN: Objection to the form.
You can answer.
THE WITNESS: As I said, I was unaware that I was a co-author on this book chapter.
BY MR. WATTS:

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Q. So you did not disclose it?
A. There was no -- there'd be no way for me to disclose it.
Q. And if we keep going in this book chapter.
Page 198. There are conclusions and future directions, and I want to visit with you about what those conclusions and those future directions are.
First it says, "A growing body of evidence suggests that dysregulation within the prenatal environment, as well as insults to the fetal brain during critical time periods of neurodevelopment or during delivery, in conjunction with genetic factors, may culminate in ASD."
Is that what's written?
A. You've read the words correctly, yeah.
Q. And do the words also say, "According to current evidence from epidemiological studies, several prenatal

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exposures, parental characteristics, and obstetrical conditions consistently emerge as potential risk factors for ASD."

Did I read that right?

A. That is what the author of the chapter wrote, yeah.

Q. And in the chapter where you were listed as a co-author, published just last year, it says, "Most notably," and then it has a list. You know, in that list is prenatal use of acetaminophen, right?

A. So among -- what the author is saying, that among the potential risk factors, they are proposing that prenatal use of acetaminophen is one possibility.

Q. And in the book chapter, which was published last year with your name as a co-author, it says, "In analyses that adjusted for confounding variables, these factors mostly remain considerably robust and statistically significant."

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Did I read that correctly?

A. You read it. I think the keyword in that sentence is "mostly." But...

Q. And on Page 199, the chapter ends with something called "Key Points." And it says, "There is strong evidence that nonheritable prenatal, perinatal, and parental events play a role in the etiology of autism spectrum disorder."

Is that what's written?

A. That's what's written.

Q. And that's written at the end of the chapter where Dr. Alexander Kolevzon is listed as a co-author of prenatal, perinatal, and parental risk factors; is that right?

A. I was listed as a co-author as a courtesy because my --

Q. And --

MS. BROWN: Well, let him finish. He's not --

MR. WATTS: I'm sorry. I was looking down.

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MS. BROWN: Go ahead. You can finish, Doctor.

THE WITNESS: So just to kind of clarify this whole chapter. There was an initial version of the Textbook of Autism Spectrum Disorders where I was an editor.

BY MR. WATTS:

Q. 2011.

A. 2011. Co-editor along with Dr. Hollander. I wrote this chapter with my colleagues Dr. Gross and Dr. Reichenberg. Subsequently, Dr. Gross, I believe, was invited to submit an updated version of the chapter that he had a student or a research associate write, and they included me as an author as a courtesy because I had written the original paper.

So there are many things in this chapter that are the opinions of the first author and perhaps other authors but not -- not of me.

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Q. So do you make it a practice of allowing your name to be used as a co-author in articles with which you disagree?

A. Absolutely not. I totally regret this, actually.

Q. And can you provide the jury with any explanation as to why it is that a CV that had 160 publications and every book chapter that your name has ever been listed on somehow miraculously did not include this book chapter that was published just last year?

MS. BROWN: I object to the form of the question as argumentive.

You can answer.

THE WITNESS: Yeah, if you're implying that I purposefully omitted this chapter from my CV for the purposes of this case, that is factually incorrect.

The reason that it's not on

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my CV is because I was not aware of it. And the reason I was not aware of it is because it was a total oversight on my part, that I take responsibility for.

BY MR. WATTS:

Q. So let's go to Exhibit 530, please.

(Document marked for identification as Exhibit Kolevzon 530.)

BY MR. WATTS:

Q. Now, the Federal Rules of Civil Procedure -- go to Page 2 -- includes a rule called Rule 26, where there is a duty to disclose and required disclosures.

Do you see that?

A. Yes.

Q. And on the next page down in 26(a)(2)(B)(iv).

MR. WATTS: Highlight it, all of that.

BY MR. WATTS:

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Q. It is a list of publications authored in the previous ten years.

Do you see that?

You know that's required, right?

MS. BROWN: I object to this line of questioning showing an expert witness the Federal Rules of Civil Procedure.

BY MR. WATTS:

Q. Go ahead, sir.

A. So, unfortunately, with chapters there's no way to cross-reference databases. When you talk about publications that are published online or through peer-review processes, you can search for it, so you can have an inclusive list.

With chapters you can't have an inclusive list. So I regret that the chapter slipped through, but I can't know something that I was not made aware of.

Q. Doctor, if we go to your CV, there's all sorts of book chapters that

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you're listed on, right?

A. Yeah.

Q. And with respect to Dr. Raz Gross, this is a medical doctor with whom you hold in the highest regard.

A. Yes, that's true.

Q. Dr. Abraham Reichenberg works with you at Mount Sinai, right?

A. Yes.

Q. A medical doctor and a researcher that you hold in the highest regard.

A. Yes.

Q. Ori Kapra, is that a doctor you respect?

A. I don't know Ori Kapra.

Q. Okay. So of the four authors that are listed, yourself, together with Reichenberg and Gross, are three individuals that you would absolutely say know exactly what they are doing from the standpoint of autism research, right?

MS. BROWN: Object to the

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form.

You can answer.

THE WITNESS: Right. Which is why I would trust Dr. Gross and Dr. Reichenberg to put appropriate things in this chapter.

BY MR. WATTS:

Q. You know --

MS. BROWN: Were you done?

THE WITNESS: Yeah.

MS. BROWN: Okay.

BY MR. WATTS:

Q. Dr. Reichenberg is an outstanding scientist in the field of autism spectrum disorder at Mount Sinai, agreed?

A. I respect Dr. Reichenberg, yes.

Q. And we can have a situation where outstanding, well-respected medical doctors just disagree; is that right?

A. I think it depends on the issue.

Q. Okay. And we'll go through

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that issue in a little bit.
But after this book, that you're listed as a co-author, said what it said with respect to acetaminophen, you had not yet been hired as a defense expert in this case; is that right?
A. Correct.
Q. You were hired on what date in this case?
A. I don't know the exact date.
Q. Okay. If I told you the first entry on your billing time records that you provided to us is December 15th of 2022, would you accept that?
A. Yes.
Q. Okay. Who first called you in this case?
A. David Cohen.
Q. Okay. And David Cohen with -- is who?
A. He's a defense attorney at Butler Snow.
Q. Okay. And he called you during the month of November, didn't he?

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A. I think the first conversation was in the month of December.
Q. Okay. And after you talked to Mr. Cohen, did you tell him that you were already in discussions with a gentleman by the name of Steve Tillery, a lawyer from Illinois?
MS. BROWN: Objection to the form of the question.
THE WITNESS: The first time --
MS. BROWN: Assumes facts. Go ahead.
THE WITNESS: The first time I spoke to Mr. Cohen, I told him that I had a conversation with Mr. Tillery, yes.
BY MR. WATTS:
Q. Okay. And after Mr. Cohen from the Butler Snow law firm, a defense lawyer in this case, contacted you, you then sent correspondence to Mr. Tillery telling him you would not be helping him

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anymore; is that right?
A. I believe the correspondence that I sent to Mr. Tillery was before I spoke to doctor -- Mr. Cohen, but...
Q. Okay. If you sent an e-mail to Mr. Tillery on the 14th and your first billing entry in this case is on the 15th of December, would that comport with your recollection?
MS. BROWN: Objection to the form. Vague.
THE WITNESS: As I recall, I had asked defense attorneys from another case that I was working on about this acetaminophen case and my discussion with the plaintiffs' attorneys. And at that point I think I had sent an e-mail to Mr. Tillery saying that I had to essentially bow out of trying to be helpful because it represented something of a conflict with another case. Not -- not related to the --

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BY MR. WATTS:
Q. And what was the other case that presented a conflict?
A. That was the Hain Celestial case.
Q. I'm sorry?
A. The Hain Celestial case.
Q. Okay. And what did it involve?
A. The one that we just discussed, the baby food causing heavy metal poisoning.
Q. Okay. And we'll get to that case in a little bit, but -- I showed you Rule 26 not to be argumentive, but you understand that if you're listed as an author on a book chapter, you have a duty to disclose it?
MS. BROWN: Object. Lacks foundation.
THE WITNESS: So, as a general rule in terms of like scientific ethics, which is what I abide by, I absolutely disclose

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everything that I'm aware of.
This was an oversight, and I regret it.
BY MR. WATTS:
Q. Okay. And, again, not beating a dead horse here, but your explanation is that it was an oversight, right?
MS. BROWN: Objection. Asked and answered.
THE WITNESS: My explanation is that this was a chapter that was written by somebody else where my name was put on it as a courtesy. And while I was aware that it was being written, I didn't have an opportunity to read it. I didn't know that it was actually being published. I didn't know what textbook it went into.
And, yes, it was an oversight.
BY MR. WATTS:

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Q. And so is today the first day that you have seen the book chapter, Exhibit 494, which you were listed a co-author of in the second edition of the Textbook of Autism Spectrum Disorders, published in March of 2022?
A. No. Defense attorneys had found it and asked me about it, I think, about a month ago, three weeks ago.
Q. Okay. And a month or three weeks ago when the defense attorneys found it and asked you about it, did you supplement your Rule 26 disclosure so that a plaintiffs' lawyer wanting to talk to you about your publications could know that you were listed as a co-author in Chapter 11 of the Textbook of Autism Spectrum Disorders, Second Edition?
A. Sorry, you have to repeat the question.
Q. Sure.
Did you supplement your Rule 26 disclosure?
A. I don't know if that was

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done or not.
Q. Okay. You know last night I got an e-mail supplementing your materials considered list as part of the Rule 26 disclosure supplementation, right?
MS. BROWN: Objection to the form. Lacks foundation.
THE WITNESS: Yeah, I don't -- I don't know whether you did or not.
BY MR. WATTS:
Q. Okay. Was there any discussion where you said, oh, that was an oversight, I need to add that to my curriculum vitae to be fair to Mr. Watts and the plaintiffs. They need to know that I'm listed as a co-author on a book chapter that says something diametrically opposed to my position in this case?
MS. BROWN: I object to the form of the question. Assumes facts. Lacks foundation.
BY MR. WATTS:

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Q. Go ahead.
A. So I think you're mischaracterizing the chapter, first of all. It doesn't totally oppose my opinions. I don't agree with everything that was written in the chapter.
I think, from my perspective, when I discovered this chapter, I do think it's important to be included now on my CV going forward, absolutely.
Q. Okay. Fair enough.
Now, you say you don't agree with everything written in the chapter with your name on it.
Do you agree that there are other researchers at outstanding medical schools that hold that same position with which you now disagree, right?
A. Can you be more specific.
Q. Sure. Let's go through a list of them.
Are you familiar with a university known as Harvard University?

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A. I am, yes.
Q. What about Mount Sinai? That's where you work, right?
A. Yes.
Q. Johns Hopkins?
A. Yes.
Q. Is that an outstanding medical research university?
A. I think it's got an excellent reputation. I think you have to judge research based on the researchers.
Q. Yale, is that a good school?
A. I'm sure Yale has some good schools within it, yes.
Q. And not subjugating Mount Sinai in anyway, but Johns Hopkins, Harvard, and Yale are all in the top ten medical schools in the United States, right?
A. I think it depends on how you define those rankings.
Q. Sure.
A. But I'm sure there's

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rankings that include them, sure.
Q. Pretty much every ranking of medical school and research facilities that exist have Johns Hopkins, Harvard, and Yale in the top ten; would you give me that?
A. I think that they are outstanding schools with good reputations, sure.
Q. Sure. And, you know, I went to a law school, University of Texas, that's in the teens, and it bothers them greatly they are not in the top 10 percent.
Mount Sinai is listed about 16 to 18, consistently, right?
A. Yeah. I went to the University of Wisconsin, which is in the 30s.
Q. And, you know --
MS. BROWN: You guys are both doing great, for the record.
BY MR. WATTS:
Q. You and I can tell Ivy

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League jokes later, but I mean, the bottom line is, is that you understand that there are researchers at your medical school, at Harvard, at Yale, and Johns Hopkins, who disagree with your position in this case?
MS. BROWN: Objection to the form. Assumes facts.
THE WITNESS: So I think that the consensus among the scientific community, at least as it relates to acetaminophen, is that it's not considered to be a risk factor and that any of the speculation that occurred in this chapter was just that, speculation.
BY MR. WATTS:
Q. Have you read the consensus statement signed by 91 scientists with respect to their concerns about the prenatal use of acetaminophen?
A. I've read the consensus statement. I've read various review

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papers.
Q. Who is the lead author --
MS. BROWN: Let him finish.
BY MR. WATTS:
Q. I'm sorry. I apologize.
Who is the lead author of the consensus statement?
A. I believe it's a person named Bauer.
Q. Okay. Do you know Dr. Bauer?
A. I do not.
Q. Who is the second author listed on the consensus statement?
A. I do not know.
Q. Do you know a Dr. Swan?
A. I do.
Q. And where does Dr. Swan work?
A. She worked at Mount Sinai. She is a collaborator of mine.
Q. Is she an outstanding autism research disorder researcher?
A. I don't think that she would

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consider herself an autism research disorder researcher.
Q. Is she a medical doctor?
A. I don't think that she is a medical doctor. I think that she is a Ph.D.
Q. Ph.D. Okay.
So we're still talking --
A. I could be wrong though.
Q. Okay. Even if she's only a Ph.D., that's a hell of an accomplishment. We still call her doctor, right?
A. Absolutely.
Q. Okay. Do you find her to be an outstanding researcher?
A. I think that she's a thoughtful scientist.
Q. She's not a hack?
MS. BROWN: Objection to the form.
THE WITNESS: I wouldn't consider Dr. Swan a hack.
BY MR. WATTS:

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Q. She's not a quack?
MS. BROWN: Objection to the form.
THE WITNESS: I wouldn't consider her a quack, either.
BY MR. WATTS:
Q. I notice that in your report you reference that clown over in England that committed, you know, medical fraud with respect to the vaccines. We're not dealing with people like that when we are talking about Drs. Bauer and Dr. Swan and folks at Harvard and Yale and Johns Hopkins, right?
A. Yeah, I don't think I would refer to any --
Q. Okay.
A. -- anybody as a clown. But Dr. Swan is a respected scientist.
Q. Okay. Dr. Reichenberg, outstanding, respected scientist, right?
A. I think that both of these people have a real commitment to trying to understand environmental causes of

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autism.
Q. Dr. Wright. What is Dr. Wright's first name, at Mount Sinai?
A. Dr. Wright?
Q. Yeah.
A. How do you spell the last name?
Q. W-R-I-G-H-T.
A. I'm not familiar with Dr. Wright.
Q. Okay. I'll show you some Mount Sinai publications. Maybe it will prompt your recollection.
A. Okay.
Q. But let's go back to medical schools for a second.
MR. WATTS: Exhibit 557, please.
(Document marked for identification as Exhibit Kolevzon 557.)
BY MR. WATTS:
Q. And this is -- just blow up the title for me. It's on Mount Sinai's

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website.
MR. WATTS: Can you blow up the title first. I'm sorry.
MS. BROWN: Oh, wow.
BY MR. WATTS:
Q. And up at the top you see Mount Sinai?
A. Yep.
Q. Okay. And it's a Mount Sinai press release from January 10, 2018, entitled "Acetaminophen Use During Pregnancy Associated With Elevated Rate of Language Delay in Girls, Mount Sinai Researchers Find."
Is that right?
A. That's what it says, yes.
Q. And in this Mount Sinai press release -- by the way, you were working at Mount Sinai at the time, right?
A. Yes.
Q. It quotes Dr. Swan. And if you can pull up --
MS. BROWN: And, Counsel, I

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don't know what to do to give him the opportunity to look at this whole thing, because the printed copy is super tiny.

MR. WATTS: Yeah. So I think that I'm going to have to plead guilty to -- I tried to buy a high-definition laser printer, and they sold me a Canon Inkjet that's just a mess.

But he has the ability to blow this up, so we'll do that on the screen. Not all of them are printed like this, but there are a few, and I apologize for that.

MS. BROWN: Okay. No worries.

Just to the extent that you need to -- you're not familiar with everything this document says, and you need to read it before you truthfully answer counsel's questions. He's going to let you do that.

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BY MR. WATTS:

Q. And the only thing I want to ask you about is the senior author of that study is Shanna Swan, Ph.D., a professor of Environmental and Public Health at the Icahn School of Medicine; is that right?

A. Yes. That's Shanna Swan.

Q. This is a person you respect, right?

A. So I can respect her science and not agree with her opinions.

Q. I didn't ask you whether you agreed with her. I said you respect her science, right?

A. I think that she is a good scientist.

Q. Okay.

MR. WATTS: Now go back to the quote.

BY MR. WATTS:

Q. She says, "Given the prevalence of prenatal acetaminophen use and the importance of language

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development, our findings, if replicated, suggest that pregnant women should limit their use of this analgesic during pregnancy."

That's what Dr. Swan said back in 2018; is that right?

MS. BROWN: I object to this line of questioning as lacking foundation.

Do you need to see the entire document to answer these questions?

THE WITNESS: So I've read this document. I'm familiar with Dr. Swan's opinions --

BY MR. WATTS:

Q. And that's what she said.

A. I just disagree with her.

Q. I know, but that's what she said, right?

A. So you've read what she said from this press release correctly.

Q. Okay. Great.

Now let's go on past Mount

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Sinai to Johns Hopkins, Exhibit 558.

(Document marked for identification as Exhibit Kolevzon 558.)

BY MR. WATTS:

Q. And the title is "Taking Tylenol During Pregnancy Associated With Elevated Risk For Autism, ADHD."

"A Johns Hopkins study analyzing umbilical cord blood samples found that newborns with the highest exposure to acetaminophen were about three times more likely to be diagnosed with ADHD and autism spectrum disorder in childhood."

Is that the title of this document that was published in November 5, 2019?

A. So you've correctly read the title. If we are going to talk about this particular opinion, I'll need to look at the actual paper that it's referencing.

Q. Okay. Well, let -- I'm just

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asking about the authors for now.
MR. WATTS: Go -- go to the quote from...
BY MR. WATTS:
Q. Do you know Xiaobin Wang?
A. I do not.
Q. Okay. Where is the Bloomberg School Department of Population, Family and Reproductive Health? Is that part of Johns Hopkins?
A. I understand it to be, yes.
Q. He says, "Our study further supports the concerns raised by previous studies- that there is a link between Tylenol use during pregnancy and increased risk of autism and ADHD."
Is that what Dr. Wang said?
MS. BROWN: Objection to the form. This lacks foundation.
THE WITNESS: You're reading quotes from press releases.
BY MR. WATTS:
Q. And my only question is, with respect to your knowledge, you had

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no information that Dr. Wang is not a highly respected researcher at one of the best medical schools in this country?
A. I can't say anything about Dr. Wang. I don't know who he is.
Q. Okay. Let's go to Yale. Exhibit 559.
(Document marked for identification as Exhibit Kolevzon 559.)
BY MR. WATTS:
Q. The title at the top says, "Scientific Team, Including YSPH" -- that's the Yale School of Public Health, right?
A. Mm-hmm.
Q. "Scientific Team, Including the Yale School of Public Health Researcher, Warn Against Use of Acetaminophen by Pregnant Women."
And that's published on September 30th of 2021; is that right?
MS. BROWN: Objection. Lacks foundation.

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THE WITNESS: This seems to be a quote from a press release, yes.
BY MR. WATTS:
Q. Okay.
MR. WATTS: And if we could go down to the paragraphs that we highlighted. Just blow those up.
BY MR. WATTS:
Q. It says, "A team of 13 scientists- including one from the Yale School of Public Health- are cautioning against the use of pain relievers with acetaminophen (also known as paracetamol) by pregnant women, citing a growing body of research that suggests the drug might alter fetal development."
MR. WATTS: Let me see the next quote.
BY MR. WATTS:
Q. I want to ask you about this person. Do you know who Zeyan Liew is?
A. I've read some of Dr. Liew's papers, yes.

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Q. Dr. Liew write -- or says, "Our lab was among the first to report a potential harmful effect of acetaminophen on fetal brain development in a large longitudinal human cohort study. It is time to take the growing body of evidence seriously and consider precautionary measures, says Zeyan Liew, Ph.D., M.P.H., an assistant professor in the Yale School of Public Health Department of Environmental Health Sciences and one of the authors of the statement"; is that right?
A. So that's what the quote says; however, if you look at Dr. Liew's paper, I wouldn't necessarily agree with those conclusions.
Q. Okay. But let me ask you, Dr. Liew is a respected researcher at the Yale School of Public Health, one of the best medical schools this country has to offer, right?
A. I don't know Dr. Liew. I can't comment on his respectability.

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Q. And so you have no negative view of his respectability, acquiring that position at one of the nation's finest medical schools?
A. I have no positive or negative view.
Q. Okay.
A. I do have a view on the results of his paper, however.
MR. WATTS: Let's go to the next paragraph.
BY MR. WATTS:
Q. It says, "The Yale School of Public Health has previously contributed to published research that raises questions about the drug's safety. This research includes a series of epidemiological studies that linked pregnancy intake of acetaminophen with an increased risk for attention deficit hyperactivity disorders, or ADHD, as well as impaired cognitive and executive function when analyzing detailed pregnancy medication intake. Data in a

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Dutch" -- "in a Danish longitudinal cohort included more than 60,000 mothers and children."
Were you involved in the research that was done by those 13 researchers, including this gentleman from the Yale School of Public Health?
A. So my research focused -- and my role, I think, in this case, is as an autism researcher and an autism expert. And this is not about autism.
Q. So let's add -- let's take out the book chapter that wasn't listed in your CV.
If you could assume, and you should, that I've read everything you've ever written, does the word "acetaminophen" show up a single time in any of the research or any of the publications that you did prior to being hired in this case?
MS. BROWN: Objection to the speech.
THE WITNESS: I can't say

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with certainty.
BY MR. WATTS:
Q. Okay. You are aware that through technology, whether it be AI or databases, we can load in everything you've ever done and do Boolean searches to look for words, right?
A. Yeah.
MS. BROWN: Objection. Lacks foundation.
BY MR. WATTS:
Q. Can you tell us whether the word "acetaminophen" shows up in any publication you ever did before this lawyer from Butler Snow called you and asked you to be an expert in this case?
MS. BROWN: I object on multiple grounds, including that it lacks foundation and it's argumentive.
BY MR. WATTS:
Q. Go ahead.
A. So I think I answered the question, which is that I cannot be

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certain. If you want me to speculate...
Q. Let's go with your recollection --
MS. BROWN: Let's let him finish, though.
BY MR. WATTS:
Q. Oh, I thought you were done. I apologize.
A. And so I think it's unlikely.
Q. Okay.
A. I have not considered acetaminophen to be a significant risk factor in autism, and I hadn't looked deeply into literature. When I had an opportunity to actually investigate the literature, it became pretty clear to me that it should not be considered a significant risk factor.
MR. WATTS: Okay. Objection. Nonresponsive.
BY MR. WATTS:
Q. Here is my question. Can you, by way of

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recollection, point me to any publication that you had ever published before being hired as an expert in this case that even referenced acetaminophen?

MS. BROWN: Objection. Asked and answered.

You can answer again.

THE WITNESS: By way of recollection, right this minute, no, I cannot.

BY MR. WATTS:

Q. Okay. Fair enough.

MR. WATTS: Let's go to Harvard, Exhibit 560.

(Document marked for identification as Exhibit Kolevzon 560.)

BY MR. WATTS:

Q. "Is a common pain reliever safe during pregnancy?"

Do you see that, sir?

A. I see what's written, yes.

MR. WATTS: And if we can go to the fourth page.

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BY MR. WATTS:

Q. It says, "Sensible steps if you're pregnant."

MS. BROWN: And, Counsel, can he have a moment to take a look at this? This one is printed in a readable format.

BY MR. WATTS:

Q. So the questions I'm going to ask you are on Page 4, about sensible steps.

MS. BROWN: But if he's never seen it before, let's give him a minute to review it so he can answer your questions.

MR. WATTS: I'm just pointing him to where we're going.

By MR. WATTS:

Q. All right. Do you know Dr. Kathryn Rexrode at Harvard?

A. No.

Q. I'm sorry?

A. No.

Q. Okay. So you don't have any

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testimony that she's anything other than an outstanding researcher --

MS. BROWN: Objection to the form.

MR. WATTS: Excuse me, I'm not done with my question. Let me -- let me ask the question and finish it and then you can object.

BY MR. WATTS:

Q. You don't have any information that Dr. Rexrode is anything but an outstanding researcher at one of the finest medical schools in this country, the Harvard-affiliated Brigham and Women's Hospital, right?

MS. BROWN: Objection to the form. Lacks foundation.

THE WITNESS: Yeah. I mean, I don't know why you're asking me about Dr. Rexrode. How is that relevant to this particular --

BY MR. WATTS:

Q. Well, it's relevant because I ask the questions and you have to

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answer them, and the judge can decide whether it's relevant.

A. Okay.

Q. And my question is, you know that Harvard is affiliated with Brigham and Women's Hospital, right?

A. Yes.

Q. It's one of the outstanding medical schools in this country?

A. It's got an excellent reputation.

Q. Okay. And in terms of this publication, the Harvard Health Publishing, it says, "Sensible steps if you are pregnant."

And the first step is, "Avoid acetaminophen during pregnancy when possible"; is that right?

MS. BROWN: Objection to the form. Lacks foundation.

THE WITNESS: Again, that's what's written here, but --

BY MR. WATTS:

Q. This says that --

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MS. BROWN: Let him finish, please, Counsel.
BY MR. WATTS:
Q. Is that what's written here?
A. So that this is a press release of sorts from a non-peer-reviewed website, I imagine. And right before it talks about sensible steps, it talks about "more research is needed."
So I agree with you that there are these things that are written. The question is what's the basis for them.
MR. WATTS: Okay.
Objection. Nonresponsive.
MR. WATTS:
Q. Under, "Sensible steps if you're pregnant," does it say, "Avoid acetaminophen during pregnancy when possible"?
A. So it's not clear on what basis this person is making this suggestion.
Q. I didn't ask you that.

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MS. BROWN: Wait. Let him finish, please, Counsel.
THE WITNESS: Those are -- those are the words on the page.
BY MR. WATTS:
Q. Okay. It also says, "'Its use should be limited to situations where it's really needed,' says Dr. Rexrode," right?
Are those words on the page?
A. It says, "Dr. Rexrode has warned patients against using NSAID drugs, such as Advil and Aleve, and suggested taking acetaminophen instead."
Q. And, keep going.
A. "Now I'll also tell you [sic] that some people -- have -- some concerns have been raised about acetaminophen use during pregnancy."
Q. "And explain that its use should be limited to situations where it's really needed," right?
A. This is what Dr. Rexrode, who I don't know who she is --

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Q. And --
MS. BROWN: Wait. Please let him finish.
THE WITNESS: -- and I don't know what basis she's making this opinion.
BY MR. WATTS:
Q. And at the bottom it says, "Minimize use. If you do need to take acetaminophen during pregnancy, take it for the shortest amount of time possible at the lowest effective dose to reduce fetal exposure. 'This advice about the lowest necessary dose for the shortest period of time is generally good counseling for all over-the-counter medication use, especially during pregnancy,' says Dr. Rexrode."
Is that the words on the paper?
A. Those are the words on the paper.
Q. Okay. Now let's go to Exhibit 561.

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(Document marked for identification as Exhibit Kolevzon 561.)
BY MR. WATTS:
Q. And this is the so-called consensus statement; is that right?
MS. BROWN: Hang on. I'm going to give him the hardcopy so he can have a minute to --
MR. WATTS: Sure.
MS. BROWN: -- refamiliarize himself before you ask the questions.
MR. WATTS: 561.
MS. BROWN: We got it.
BY MR. WATTS:
Q. Is this the one where Dr. Bauer is the first author and Shanna Swan is second?
A. Yes.
Q. And in the abstract does it say, "We recommend that pregnant women should be cautioned at the beginning of pregnancy to," and it says, "forego APAP

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unless it is medically indicated; consult with a physician or pharmacist if they are uncertain whether its use is indicated and before using on a long-term basis; and minimize exposure by using the lowest effective dose for the shortest possible time."

Is that what it says?

A. I think this group of authors is providing their opinion on the use of acetaminophen and urging caution.

Q. And is that what it says?

A. It's what it says.

Q. Okay.

A. But I don't think that's commonly accepted in the scientific community.

Q. Now, let's talk about that for a second.

MR. WATTS: Go to Exhibit 562.

(Document marked for identification as Exhibit Kolevzon 562.)

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BY MR. WATTS:

Q. And this is a supplementary information to the consensus statement. It's entitled "Paracetamol Use During Pregnancy: A Call for Precautionary Action."

Do you see that?

MS. BROWN: Hang on a second. I don't -- I just want to get us the hardcopy, and I don't see it.

Here we go.

BY MR. WATTS:

Q. Have you gone through and studied these 91 doctors that signed off on the consensus statement?

MS. BROWN: Objection. Vague.

THE WITNESS: Can you clarify the question.

BY MR. WATTS:

Q. Sure.

Did -- did you go through and check who they are, see their

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reputations?

MS. BROWN: Objection.

THE WITNESS: No.

BY MR. WATTS:

Q. Okay. Let's go to Page 2, the supplementary box, the signatories.

Do you see, beginning on Number 1 -- we can flip through the pages, and it will go from 1 to 91 -- as we go to Page 2, Page 3, Page 4. Stop at Page 5.

And it keeps going to 91, but let me ask you about Number 41.

Do you know who Martha Herbert is?

A. 41.

MS. BROWN: Just a minute while he gets there.

THE WITNESS: Martha Herbert?

BY MR. WATTS:

Q. Herbert? I'm sorry.

A. I do not.

Q. Is Massachusetts General

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Hospital an outstanding medical facility in this country?

A. MGH has a good reputation, yes.

Q. Stephen Schultz, do you know Stephen Schultz, Number 42?

A. I don't believe I know Stephen Schultz, no.

Q. Now, I don't want to get crossways with you, but this is a gentleman at the University of Texas Health Science Center in San Antonio, Texas, and we are pretty proud of him.

Do you know him?

A. This is Dr. Schultz again?

Q. Yeah.

A. No, I still don't know him.

Q. Dang.

A. Sorry.

Q. Okay. How about Number 48. Do you know Dr. Ritz at UCLA?

A. No.

Q. Is UCLA considered one of the top medical facilities in this

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country?
A. UCLA has some excellent programs, yes.
Q. Okay. Let's go to 57.
A. Yeah.
Q. Do you know Dr. Bergink?
A. I do.
Q. She's one of your colleagues at Mount Sinai, right?
A. She is.
Q. Dr. -- Number 80, Shanna Swan?
A. I know Dr. Swan, yes.
Q. One of your colleagues at Mount Sinai, right?
A. She works at Mount Sinai, yes.
Q. And then if we go to 82, we've got Dr. Liew at Yale and Dr. Hugh Taylor. Do you know Dr. Taylor at Yale?
A. I don't know either one of these personally.
Q. Okay. What about Number 91, Dr. David Møbjerg Kristensen?

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A. I don't know Dr. Kristensen personally, no.
Q. Has he taught at Icahn on an adjunct professor basis, do you know?
MS. BROWN: Objection to the form. Lacks foundation.
THE WITNESS: He may or may not have. I am not aware.
BY MR. WATTS:
Q. Okay. And if we go to Page 12, "Supplementary Table 1: Reproductive [sic] Epidemiology."
They list the epidemiology, right?
A. Page 11, yeah.
Q. Supplementary Table 2 on Page 14. They list "Reproduction Experimental Studies," right?
A. That's what Table 2 says, yes.
Q. Page 20, Supplemental Table 3, they list "Neurodevelopmental Epidemiological" -- or "Epidemiology Cohort Studies," right?

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A. Yep. There are studies listed here. That's correct.
Q. Now, if we go to Page 32, there's a "Supplementary Table 4: Neurotoxicity Experimental Studies"; is that right?
MS. BROWN: Hang on just a sec while he gets there.
THE WITNESS: These look to be model studies, animals, yeah.
BY MR. WATTS:
Q. If we go to Page 37, Supplementary Table 5 lists review articles, right?
A. Sorry, which page?
Q. Page 37.
A. There are some review articles in this table, yes.
Q. And if you go to Page 50, they begin the inclusion of references. In between 50 and 61, there are 121 different references that they cite to; is that right?
A. There appear to be

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121 references.
Q. Now, let me go back to Yale for a second, Exhibit 563.
(Document marked for identification as Exhibit Kolevzon 563.)
BY MR. WATTS:
Q. This is dated the spring of 2022. Came out about the same time as your book chapter.
"Yale School of Public Health Research Identifies Pregnancy Risks Associated With Acetaminophen Use."
MS. BROWN: Sorry, what -- what exhibit is this?
MR. WATTS: 563.
BY MR. WATTS:
Q. Did you see this when it came out?
MS. BROWN: Hang on a second while we find it.
Did we mark this already?
MR. WATTS: Nope.
MS. BROWN: Okay.

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BY MR. WATTS:
Q. Do you recall seeing this when it came out?
A. I just need to look at the article.
MS. BROWN: Just -- just a second. I'll give it to him. It's a little bit hard to read.
Mr. Watts can probably put it up on the Elmo if you need it expanded --
THE WITNESS: I just want to -- want to see what article they are referencing.
BY MR. WATTS:
Q. What I want to ask you about is the third paragraph from the bottom. It says, "In another study."
MS. BROWN: But, Counsel, just because he's never seen this before --
MR. WATTS: I know. I know.
MS. BROWN: -- if we can just give him a minute to

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familiarize --
BY MR. WATTS:
Q. I know. I'm just pointing you to where I want to ask you about.
MS. BROWN: Thanks.
MR. WATTS: Just blow up "In another study," if you would.
MS. BROWN: Well, give him a second to read this.
THE WITNESS: Okay.
MR. WATTS: Now blow up that article. "In another study."
There you go.
BY MR. WATTS:
Q. All right. Here is my question.
It references another study where a Yale student "used human pluripotent stem cells, RNA sequencing, and metabolomics to identify cellular mechanisms that may be involved in the development of autism spectrum disorder. After exposing the stem cells in culture to clinically relevant doses of

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acetaminophen for six days, Furnary found that acetaminophen to elicit the same gene expression patterns and metabolic behaviors in the cultures as those known to be associated with autism spectrum disorder."
Do you see that, sir?
A. Yeah, I see that.
Q. Here's my question:
Can you tell the ladies and gentlemen of the jury what the phrase "gene expression" means to you?
A. So gene expression is, essentially, whether the proteins that are translated from the DNA are upregulated or downregulated.
Q. Okay. And when this says that "the same gene expression patterns and metabolic behaviors in the cultures of acetaminophen were those known to be associated with autism spectrum disorder," have you seen this work?
MS. BROWN: Objection to form. Vague.

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You can answer if you understand.
THE WITNESS: That's the -- that's the end of the question?
BY MR. WATTS:
Q. Yeah.
A. Oh.
So I'm not sure I feel comfortable commenting on the study without looking at the actual study. So this is just an excerpt.
Q. Does this ring a bell to you at all as something that you recall looking at?
A. I'm not sure I understand the question.
Q. Sure.
This -- this gene expression work that was done at Yale, does it ring a bell?
A. As it relates to what specifically?
Q. Acetaminophen and autism spectrum disorder.

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MS. BROWN: Objection to the form.
THE WITNESS: So I'm aware of studies that have looked at proxies for gene expression.
BY MR. WATTS:
Q. Okay. Now, have you done any studies that used proxies of gene expression to compare what the gene expression patterns were for acetaminophen versus what you see in autism spectrum disorder?
A. I myself do not do gene expression studies.
Q. Fair enough. All right.
MR. WATTS: Let's go to Exhibit 403.
(Document marked for identification as Exhibit Kolevzon 403.)
BY MR. WATTS:
Q. And this is your report. You're probably going to need it most of the rest of the day, so just kind of --

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don't put this one up.
A. Okay.
Q. Is Exhibit 403 a true and correct copy of the expert report that you provided in this case?
MS. BROWN: And just at least take a moment to flip through it, please.
THE WITNESS: Yeah, it looks to be.
BY MR. WATTS:
Q. Now, you've also gave a report in a case called Daniels-Feasel; is that right?
A. Yes.
Q. And that's Exhibit 479. If you could pull that one out as well.
(Document marked for identification as Exhibit Kolevzon 479.)
BY MR. WATTS:
Q. Is the date of this report November 8th of 2018?
A. That's what it says on the

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top of the report, yes.
Q. Now, if you could, you see the file number that says Page 2 of 94, and then it goes up one per page?
A. Yes.
Q. Okay. Go to Page 9 of 94 for a second.
MR. WATTS: Now, if you would blow up the first five lines, Erik, of III (a)?
BY MR. WATTS:
Q. Now, you described autism disorder first being described by Leo Kanner in 1943.
Second paragraph, you said, "The prevalence of ASD has increased dramatically over the past five decades in the United States"; is that right?
A. That's what it says, yes.
Q. All right. Now, I want to talk to you about prevalence in some detail for a little bit.
Between 1997 and 2007, do the prevalence rates of autism increase

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in the United States?
A. The prevalence increased. I'm not sure the incidence did.
Q. I'm asking about prevalence rates right now, okay?
A. So according to prevalence-based studies, depending on the methods, most rates increased.
Q. And let me show you Exhibit 414 where you've said this, just real briefly.
(Document marked for identification as Exhibit Kolevzon 414.)
BY MR. WATTS:
Q. And we'll talk about prevalence versus incidence here in a second.
Did you write an article in 2007 entitled, "Prenatal and Perinatal Risk Factors for Autism: A review and integration of findings," with Dr. Gross and Dr. Reichenberg?
A. I did.

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Q. And on Page 326 there's a sentence in there that says, "Prevalence rates of both autism and autism spectrum disorders (ASDs) may have increased markedly in the past decade"; is that right?

MS. BROWN: Can we give him a second to find the page. Page 3 of the article.

MR. WATTS: No, I said Page 326, which is the first page.

THE WITNESS: Are you referring to the abstract or the --

MS. BROWN: Right here.

THE WITNESS: Ah, okay. That's convenient.

Yes, I think it's inarguable that prevalence rates have increased.

BY MR. WATTS:

Q. Okay. And then in the next column it says, "Although this increase may be artifactual to some degree, it may

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also reflect a true increase in the incidence of ASD and implicates an important role of environmental causes."

Did I read that correctly?

A. You read it correctly. And it reflects the idea that there are some nonheritable causes and that they're important to try to identify.

Q. And I think your point is that if you have this dramatic increase of autism spectrum disorder cases, our genes don't change that fact. So if there is, in fact, a true increase, it has to be explained by something other than genetics; is that right?

MS. BROWN: Objection. Lacks foundation.

THE WITNESS: So my opinion about the true increase and prevalence relates more to other factors, like changing diagnostic criteria, younger age of diagnosis --

BY MR. WATTS:

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Q. And what I'm --

MS. BROWN: Let him finish. Let him finish.

BY MR. WATTS:

Q. Yeah, go ahead.

MR. WATTS: We're going to get to those.

MS. BROWN: I know. But let him at least answer your question and then you can follow up.

Go ahead.

THE WITNESS: You had said that -- something to the effect of because there's an increase in prevalence, that must mean environmental factors, and I don't think that that's true --

BY MR. WATTS:

Q. I actually said --

MS. BROWN: Wait. Wait. Please, Mr. Watts, let him finish.

MR. WATTS: I'm sorry.

MS. BROWN: Go ahead, sir.

MR. WATTS: I actually said,

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"If there is a true increase."

MS. BROWN: I know, but he's got to at least finish and then you can follow up.

BY MR. WATTS:

Q. Go ahead. Go ahead. I didn't mean to cut you off. Go ahead.

A. Okay.

So the fact that there's an increase in prevalence does not necessarily reflect the presence of environmental risk factors.

Q. Could be artifactual, but if it's not artifactual, it has to be environmental. Agreed?

A. I don't think it has to be anything. I think that there could be other factors that we are, as of yet, unaware of.

Q. Okay. So let's talk about whether it's artifactual.

But before we go there, let's -- let's talk about the increase of prevalence over time.

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Let me show you Exhibit 417.
(Document marked for identification as Exhibit Kolevzon 417.)
BY MR. WATTS:
Q. Do you know who Dr. Hertz-Picciotto is?
A. I don't know her personally.
Q. But you published with her?
A. I may have been on papers with her, but I don't know her.
Q. Okay. And she did a lot of work with respect to the increase in prevalence in -- shown in certain databases out in California; is that right?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: I know that she works with some California databases, yes.
BY MR. WATTS:
Q. And if you go to Page 3 of 20.

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A. Hold on. Hold on one second. Let me just look at the abstract.
MS. BROWN: I think we only have a page.
If this is Exhibit 417 -- are we looking at the same thing?
THE WITNESS: "The Rise in Autism and the Role of Age at Diagnosis."
MS. BROWN: Is that it? Our hardcopy just has one page.
MR. WATTS: There's one page in your folder?
THE WITNESS: Two pages.
MS. BROWN: It looks -- Mr. Watts, it looks like it's just the abstract.
MR. WATTS: That's odd. Here, let me give you mine.
MS. BROWN: Okay.
MR. WATTS: Will you make sure that we substitute the right one in there?

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I apologize.
THE WITNESS: Thanks.
BY MR. WATTS:
Q. Just go to Page 3 of 20, and I'm going to ask you about the data. And then you and I are going to talk about the different reasons that you think it's artifactual.
A. Yeah, okay. I just want to look at the way the study was done.
MS. BROWN: Okay. Give us a second to organize our exhibits.
THE WITNESS: I'm going to need more time with this paper. I mean, I have not seen this paper before. So if you are going to ask me questions about it, I need more time to review it.
BY MR. WATTS:
Q. Well, let me just ask you -- and I want to be fair to you.
This is something you haven't seen before?
A. Correct.

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Q. Okay. And then last question, and then we'll go on, because I don't want to talk to you about something that you haven't seen.
Just on the results on Page 3 of 20. Do you see how it says, "Autism incidence in children rose throughout the period. Cumulative incidence to five years of age per 10,000 births rose from 6.2 for 1990 births to 42.5 for 2001 births"?
MS. BROWN: And I'll just object. The witness asked for more time with a paper he's never seen.
So if you want to ask him about the paper, that's fine. He just needs time to look at it.
BY MR. WATTS:
Q. I'm just asking about this one sentence.
Do you see that, sir?
MS. BROWN: Well, I object as lacking foundation.

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BY MR. WATTS:
Q. Go ahead.
A. So I see that that's what's written on the page, but I have no way to evaluate it.
Q. And the way I want to ask the question is this:
The difference between 6.2 and 42.5, how would you express that change in rate?
A. Again, I think it --
MS. BROWN: Object. It lacks foundation. I object.
He's never seen this article before and he asked for more time to answer your question.
So I object.
MR. WATTS: You can have a running objection.
BY MR. WATTS:
Q. Go ahead.
A. I seek to understand the methods of the paper. I need to understand the cohort, the rigor, the

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science.
Q. Okay. And my question is, what's the difference between 6.2 and 42.5, mathematically?
A. Well, if you want a mathematical --
Q. Yeah.
A. It's times seven.
Q. Okay. So seven-times increase, right?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: That's the quantitative difference, but I don't know how that relates or how that's relevant to the incidence of autism.
BY MR. WATTS:
Q. Okay. So the reason I ask about this is this seven-time difference, quantitatively, between 1990 and 2001, in your report, you have four or five artifactual reasons that you think that difference exists, right?

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MS. BROWN: I object as lacking foundation.
THE WITNESS: I think the consensus in the scientific community is the prevalence of autism is increasing. And the reason it is increasing is because of a number of different factors.
But, yes, you would probably label those artifactual.
MR. WATTS: Okay. Objection. Nonresponsive.
BY MR. WATTS:
Q. I'm asking about you and your report.
And in your report you suggest five artifactual reasons that explain whatever the rate increase is, right?
A. My report reflects on the reasons that the prevalence rates have gone up.
Q. Okay.
A. And my opinion is that there

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isn't a true increase in the -- in the incidence.
And if there is one paper that says that there is, I need to evaluate that paper more, in order to comment.
Q. I understand.
Now I want to talk to you about the incidence increase over the last decade.
Can I have that back? And then I'll get you a copy during the break.
MR. WATTS: Will you go get that?
MS. BROWN: Do you want to take a break now so we can get him a copy before you start --
MR. WATTS: We're going to go on to something else. Let me finish this section and we'll take a break.
MS. BROWN: Okay. And I'll just continue to object to the

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extent the witness needs a copy --
MR. WATTS: Yes.
MS. BROWN: -- and time to review before he answers questions about a paper he's said he has never seen before.
MR. WATTS: Let's go to one you wrote. Exhibit 422.
(Document marked for identification as Exhibit Kolevzon 422.)
THE WITNESS: Ah, first edition.
BY MR. WATTS:
Q. There you go.
You wrote this in 2011, right?
A. I think this was published in 2011. I probably wrote it before then, but...
Q. Go to Page 568.
A. 568.
Q. It's in the very back.
By the way, just for the

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record, there are three book chapters that you included in here. I'm going to the third one.
You wrote three. So 568 is towards the back. Chapter 47, "Future Directions."
A. "Future Directions." Okay.
Q. And by the way, if we go to 567 --
MR. WATTS: Erik, the previous page, please.
BY MR. WATTS:
Q. This is a book chapter that you wrote with Eric Hollander. Did you work with Dr. Hollander at Mount Sinai for a number of years?
A. I did, yes.
Q. How long did you work together with Eric Hollander?
A. I think Eric -- I was in Mount Sinai from 2000. Eric was there well before me, and I think he was there until 2009. So about nine years.
Q. Did you work under him?

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A. I did.
Q. Okay. Was he your boss between 2000 and 2009?
A. No, he was my boss between 2007 and 2009.
Q. Okay. Is he a medical doctor that you respect?
A. I respect Dr. Hollander.
Q. Okay. He is a fine researcher in the field of autism spectrum disorders, right?
A. Dr. Hollander does a lot of research in autism. I don't always agree with his conclusions, but he does a lot of research.
Q. And, again, I'm not trying to get you to say something to get you in trouble with your buddies.
But in science, you can have two different outstanding researchers who both employ sound science and can come up with this different opinions, right?
MS. BROWN: Well, I object as vague.

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Are you talking about generally or are you talking about related to his opinions in this case?
BY MR. WATTS:
Q. Go ahead.
A. I think --
MS. BROWN: Same objection. Go ahead.
THE WITNESS: I think it depends on what the issue is and what the data show.
BY MR. WATTS:
Q. Okay. But my point is, is that Dr. Kolevzon is an autism spectrum disorder researcher for whom you hold the highest regard?
MS. BROWN: He is Dr. Kolevzon.
THE WITNESS: Dr. Kolevzon? I am Dr. Kolevzon.
BY MR. WATTS:
Q. Well, I knew that one was true. I meant Dr. --

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MS. BROWN: So he is very, very well respected.
THE WITNESS: I like to think I'm more humble than that, but...
BY MR. WATTS:
Q. You know, if we can't laugh about moments like that, you can't laugh.
Other than holding yourself in high regard, you hold Dr. Hollander in high regard as an outstanding researcher in the -- in the field of autism spectrum disorder?
MS. BROWN: Same objection.
BY MR. WATTS:
Q. Go ahead.
A. I respect Dr. Hollander as a well-intentioned, thoughtful scientist.
Q. How about Dr. Coyle?
A. I respect Dr. Coyle as a well-intentioned, thoughtful scientist.
Q. Okay. And if we go to the next page, under "Prevalence," the three of you write, "The prevalence of autism

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continues to grow. The most recent Center for Disease Control and Prevention estimates suggest 1 in 110 persons have an ASD, which is higher than the earlier estimates of 1 in 150 affected individuals."
Did I read that right?
A. Yes.
Q. Let's move forward to Exhibit 479, which is your report in the Daniels-Feasel case.
Page 9 of 94. Now we are in November of 2018?
And by here, in Paragraph III (a), you say the prevalence rate is 1 in 59; is that right?
A. I'm quoting the CDC.
Q. Okay. And then let's go to 2021. Exhibit 491.
(Document marked for identification as Exhibit Kolevzon 491.)
BY MR. WATTS:
Q. Do you recall a paper you

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wrote with Dr. Katz?
MS. BROWN: Hang on. 491?
MR. WATTS: Yes, ma'am.
BY MR. WATTS:
Q. Julia Katz?
MS. BROWN: Hang on. We're getting it out.
BY MR. WATTS:
Q. Is Julia Katz an autism researcher you respect?
A. Julia was a resident in general psychiatry. So she is not an autism researcher, no. She did autism research in this case.
Q. Yeah. I mean, we all start off as a resident, right?
A. Basically. I mean...
Q. Some of us are such outstanding researchers we can write books during our residency, can't we?
A. Some of us.
Q. Abraham Reichenberg was not in his residency when he co-authored this article with you, right?

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A. Dr. Reichenberg is a -- is an epidemiologist and Ph.D., so he doesn't do a residency.
Q. Okay. Go to Page 2. The introduction.
The three of you, with respect to prevalence at this time, and we're now in March of 2021, say, "The prevalence of ASD has been increasing in recent decades and current estimates from the Center for Disease Control (CDC) suggest that 1 in 54 children in the U.S. aged eight years has ASD."
Did I read that right?
A. Yeah, those were the CDC rates at the time that we published this.
Q. And then lastly, Exhibit 568.
(Document marked for identification as Exhibit Kolevzon 568.)
BY MR. WATTS:
Q. I saw in your supplemental disclosure last night that you read the

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transcript of Wendy Chung's deposition.
A. I did, yeah.
Q. Let me show you Exhibit 568, which is a PowerPoint that she published on April 25th of 2023, entitled, "SPARK and the Future of Autism Research."
MS. BROWN: Yeah. I'll just object to the form as inconsistent with her testimony about this.
Go ahead.
MR. WATTS: Okay. And I will stipulate that what she testified to is inconsistent with what she's written.
I'm kidding. Let's go on.
MS. BROWN: I get the sense that you guys think that. I think it's --
BY MR. WATTS:
Q. By the way, Doctor, Alli and I are good friends, and we can joust. It's nothing personal.
A. I was told not to do any humor, so...

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Q. Try not to be funny.
Let's go to Page 7, please.
MS. BROWN: Which in and of itself is hilarious.
All right. We're focusing on Exhibit 560?
MR. WATTS: 8.
MS. BROWN: 8.
BY MR. WATTS:
Q. And in April 2023 Dr. Chung says, "The prevalence of autism, according to 2020 data, has increased to 1 in 36 eight-year-old children."
Do you see that?
A. I see what's written on the screen, yes.
Q. All right. Now, let me show you Exhibit 543, just for a second.
MS. BROWN: And this is the last one?
MR. WATTS: We'll take our break after this.
MS. BROWN: Great.
(Document marked for

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identification as Exhibit Kolevzon 543.)
BY MR. WATTS:
Q. I took the studies we just went through, between 2011 and 2023, and without making you retread all these numbers, I'm sure somebody at the fine firm where Alli comes from will check my math.
Can we agree that the rate of reported ASD from 2011 to 2023 has increased during those 12-year period?
MS. BROWN: Okay. And I'll object.
Can you tell us what this is.
Did you make this, Mr. Watts?
MR. WATTS: I did.
MS. BROWN: Okay. So I'm going to object as lacking foundation to this un-cited, lawyer-created chart.
BY MR. WATTS:

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Q. Just the ones we just read in show an increase between 2011 and 2023 in the rate of ASD prevalence reported over that time, right?
A. So over this period of time, there have been dramatic changes that have contributed to the increase in prevalence of autism.
Q. Okay.
A. The CDC, whose rates you've quoted, uses a methodology that, by itself, inflates the rates. And so if you're asking me whether or not rates have gone up, prevalence rates, over the last 12 years, the answer is yes, undoubtedly.
Q. Okay. With respect to the issue of whether the rates of reported ASD have gone up, we can agree that that has happened. You just say there are reasons for it that are artifactual, fair?
A. Fair.
MR. WATTS: Okay. We're

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going to take our break and then we're going to talk about those reasons.

THE WITNESS: Great.

MR. WATTS: All right.

THE VIDEOGRAPHER: The time right now is 9:40 a.m. We are off the record.

(Short break.)

THE VIDEOGRAPHER: The time right now is 10:00 a.m. We're back on the record.

BY MR. WATTS:

Q. I want to go back to the Katz paper that you co-authored in March of 2021, Exhibit 491.

And the purpose of the review says, "Given the ongoing rise in prevalence of autism spectrum disorder (ASD)" -- and then you go on to say, "There is an urgent need to identify modifiable risk factors for ASD."

What is a modifiable risk factor?

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A. It's one where you can manipulate the environment in some way or you can provide some sort of medication to either prevent or alleviate symptoms.

Q. So a modifiable risk factor, by definition, relates to environmental causes of ASD?

A. Not by definition and not exclusively. But environmental risk factors could be considered a modifiable risk factor, yes.

Q. Okay. Now let's go into the reasons for the increase of the prevalence, and I want to refer you to your report, Exhibit 403, Pages 16 and 17, and Paragraph 39. Can you pull that up.

A. Sorry, page? Page what?

Q. 16 and 17, Paragraph 39.

Okay. Now, you acknowledge that prevalence rates have gone up, but you say, "It's unlikely the true incidence of ASD is increasing. This discrepancy is due to a combination of

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factors that broadly include A, B, C, D, and E."

Do you see that, sir?

A. I do.

MR. WATTS: Now, if you would put up Exhibit 545, just for a second, Erik.

(Document marked for identification as Exhibit Kolevzon 545.)

BY MR. WATTS:

Q. What I've done is I've taken A, B, C, D, and E and put it into an Excel chart.

And here is my question:

First of all, under the explanation, did I correctly transpose from your report the five reasons that you posit explain the increase in the reported prevalence rate increase of ASD?

A. Yeah. I think the only clarification I would make is it's not about calculating the prevalence rate, it's about the ascertainment.

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Q. Okay. Now, here is my question:

Each of these five factors that you listed in your report, for example, E, "changes in law and society," you reference something that happened back in 1991, right?

A. Yes.

MR. WATTS: Okay. So, Erik, could you write "1991" under date.

BY MR. WATTS:

Q. Now, in C, for example, "younger age of diagnosis," you report this is something that happened back in 2007, right?

A. There are many factors that contributed to younger age of diagnosis. But in 2007 is when the recommendation from the American Academy of Pediatrics came out.

MR. WATTS: Okay. Type in "2007" under B, Erik.

TRIAL TECH: I'm sorry, under D or?

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MR. WATTS: B.
THE WITNESS: No, no.
MR. WATTS: Apologies. Type in "2007" under C.
BY MR. WATTS:
Q. Now, under D, the "improvements in diagnostic ascertainment," you reference a behavioral tool called ADOS in your Daniels-Feasel report and then Autism Diagnostic Interview-Revised, an ADI-R, in that same report.
You know what those are?
A. I do.
Q. And here is my question.
From the standpoint of improvements of diagnostic ascertainment, the ADOS was created by Catherine Lord, Michael Rutter, Pamela DiLavore, and Susan Risi back in 1989, right?
A. That's correct.
Q. And the ADI-R was developed by Rutter, Ann Lecouteur, and Catherine Lord in 2003; is that right?

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A. Both those things are correct. But neither one of those things would I necessarily attribute to the improvement in diagnostic ascertainment.
Q. Well, let's go and see what you said in Exhibit 479, which was your Daniels-Feasel report.
Go to Page 10 of 94.
MR. WATTS: You don't need to type Exhibit 479.
MS. BROWN: Hang on. Do we have it? Did we already look at this one?
THE WITNESS: Yeah.
MS. BROWN: Okay. Let's just find it.
MR. WATTS: 794. III (b).
BY MR. WATTS:
Q. And three lines down you say, "Finally, behavior assessment tools, such as the Autism Diagnostic Observation Schedule (ADOS) and the Autism Diagnostic Interview-Revised have improved our ability to detect autism."

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Is that what you said in that report?
A. It's true that those are better measures to detect autism, but that's not necessarily --
Q. And those better measures happened in 1989 and 2003, right?
A. Right. But the ability to detect autism and ascertainment methods can sometimes be different.
Q. Okay. With respect to this report, those are the two things that you referenced, right?
A. So in this report, I'm talking about two different tools that were developed that improved our ability to detect autism.
Q. That happened in 1989 and 2003.
A. Well, there have been multiple revisions over time that have improved the tools. But they were originally developed earlier, yes.
Q. Okay.

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MR. WATTS: Now go back to Exhibit 545, Erik.
BY MR. WATTS:
Q. And so 1989 and 2003.
The expansion of ASD diagnostic criteria. Are you talking about the iterations of the DSM?
A. I am.
Q. Okay. And if we look at the iterations of the DSM, the DSM-III was published what year?
A. 1987.
Q. Are you sure that wasn't the DSM-III-Revised?
A. Oh, correct. Sorry.
Q. 1980 -- let me just kind of lead you through this.
1980 was the DSM-III, right?
A. Correct.
Q. 1987 was the DSM-III-Revised.
A. R, yeah.
Q. 1994 was the DSM-IV?
A. Yes.

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Q. And 2013 was the DSM-V?
A. Correct.
Q. Okay. So when we talk about B, that would be the various iterations of the DSM, the Diagnostic and Statistical Manual, right?
A. Correct.
Q. All right. So that happened between 1980 and 2013, right?
A. Yes. The most dramatic of which probably was 2013.
Q. All right. Now, with respect to A, the "methodological issues in calculating prevalence rates," your report references a study by Avchen, right?
A. Yes.
Q. And basically what you're saying is that because of the data found in Avchen, the calculated prevalence rate may be inaccurate; is that right?
A. So I need -- I need to make a couple of comments about this.
Q. First of all, is that right,

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and then you can make your comment.
A. So Avchen is one paper which supports my opinion.
Q. It's one you cited, right?
A. I cite it.
I have 20 years of experience that support my opinion.
The consensus within the scientific community supports my opinion.
But more importantly, I don't think it's fair to assign a specific date to these dynamic issues that have been unfolding for the last 50 years.
Q. Avchen looked at 177 children in Atlanta who were born in 1997 and went to public schools in 2005, right?
A. Yeah.
Q. Did you cite to any other specific studies referencing methodological issues in calculating prevalence rates, other than Avchen?
A. It's commonly accepted

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that --
MS. BROWN: Well, let him finish and then you can follow up.
Go ahead.
THE WITNESS: It's commonly accepted that the diagnosis of autism requires a clinician to observe a child, and that requirement is not embedded within the ADDM, the CDC criteria.
And when Avchen went and looked at a subset of people who had been diagnosed, according to the CDC criteria, found that a certain percentage did not meet criteria.
BY MR. WATTS:
Q. Okay.
A. And that's consistent across clinics and that's consistent across the literature. And that -- that's just the way that the scientific community accepts the increase in prevalence.
Q. What was my question?

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A. Your question was whether -- well, why don't you repeat your question.
Q. Sure.
Did you cite to any other studies in support of your proposition other than Avchen?
A. I provided Avchen as a reference.
Q. As an example?
A. But not to imply that that's the only reference --
Q. Sure.
A. -- or that that's the totality of the literature that I'm relying on.
Q. Sure. But you understand when you write a report and you drop footnotes, I'm going to read those footnotes.
A. Yeah.
MS. BROWN: Objection.
BY MR. WATTS:
Q. And if you cite one example, and I go read it and can joust about that

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one example. And then you come in and say, oh, it's conclusive, the scientific community knows this, and like that, that doesn't provide me with any other studies that back it up, does it?

MS. BROWN: No. I object. That lacks foundation. It's argumentive, and it's also false.

BY MR. WATTS:

Q. Go ahead.

A. I think it's clear, based on my record and my experience and my publications, that I'm considered an expert. I think it's clear that I can be representing the scientific community under what the consensus is as it relates to these issues. And I think it's clear that, in general, these are the themes that explain the increase in prevalence rates.

Q. Okay. So was Avchen ever replicated?

A. I think there was one other study, but I can't recall it off the top

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of my head.

Q. Did Avchen state, we have used rules to resolve discordance between ASD diagnostic measures that have not been studied or replicated in published research?

A. If you want to discuss the Avchen article at length, I need to pull it out and review it.

MR. WATTS: Okay. Exhibit 420, Page 235.

(Document marked for identification as Exhibit Kolevzon 420.)

BY MR. WATTS:

Q. The question is did they state that.

First column, first full paragraph, fourth line.

MR. WATTS: Blow it up. No, first full paragraph.

"Several limitations in this study deserve consideration."

BY MR. WATTS:

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Q. Did they state, we used rules to resolve discordance between ASD diagnostic measures that have not been studied or replicated in published research?

MS. BROWN: Where are we?

THE WITNESS: Yeah, I'm not sure where we are.

MS. BROWN: Can you show us where we are? It's not matching up --

MR. WATTS: Sure. Erik highlighted it.

MS. BROWN: I know, just we want to match it up --

THE WITNESS: What page?

BY MR. WATTS:

Q. It's on 235. First column, first full paragraph. And it's being highlighted on the big screen right in front of you.

A. 235.

MS. BROWN: I think we might have a different article printed.

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Do you see this?

THE WITNESS: It's -- I don't have -- I mean, 235 is Page 1. It doesn't --

MS. BROWN: Yeah, we have a different copy printed here, you guys.

What is the date?

MR. WATTS: Erik, pull up the paragraph, please.

TRIAL TECH: They're trying to -- I'm trying to help them --

MR. WATTS: I want you to blow up the paragraph that --

MS. BROWN: I think we just need to get coordinated, because we're looking at something totally different.

MR. WATTS: Okay. It sounds like we had a bad copy job, so I apologize for that.

MS. BROWN: No worries.

BY MR. WATTS:

Q. Yeah. Can you see the

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screen, sir?
A. Yeah.
Q. Can you see the words, "We use rules to resolve discordance between ASD diagnostic measures that have not been studied or replicated in published research"?
MS. BROWN: And, Mr. Watts, I don't mean to be difficult, but we don't have this document in front of us, and I think he needs to see it just to be able to accurately answer your questions.
So can we just resolve that?
MR. WATTS: Take -- take my copy too.
MS. BROWN: All right.
MR. WATTS: I don't know what happened to your copy. I apologize.
MS. BROWN: All right. No worries. Thanks very much. Thanks very much. Thank you.
BY MR. WATTS:

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Q. 235.
A. Oh, it's a different pagination altogether. Okay.
MS. BROWN: Okay. All right.
BY MR. WATTS:
Q. Do you see the words --
A. I just -- I need to understand the context. I'm sorry. I appreciate these are limitations that the authors are citing. I just want to understand in what context.
Q. Okay. And before we get to the full context, do the words, "We used the rules to resolve discordance between ASD diagnostic measures that have not been studied or replicated in published research," is that in there?
A. Those are the words that are written on the page.
Q. All right.
MS. BROWN: And we can resolve this on a break, but both of the copies seem to go up to 236

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and then back down to 233. So I don't --
MR. WATTS: Look, I would assume that whoever they sent out the copies to screwed up and used the digital version that I've given you.
But if there's a problem, let me know, and I'll give you mine just like I did. I don't have a --
MS. BROWN: Yours is printed with the same issue. So I just want to -- I just want to make sure we have the complete study --
MR. WATTS: Of course.
MS. BROWN: -- and Dr. Kolevzon has a chance to refresh on it so we can accurately answer your questions.
So if you need a minute with whatever we have in front of us, please take a minute. Let's make sure we're all on the same page

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before you answer.
THE WITNESS: Do you have additional questions?
BY MR. WATTS:
Q. No, that was the question.
And then my next question is, in Avchen, did they have misclassified children who were incorrectly transferred from case to non-case status as well?
A. So, again, if we want to talk about Avchen in detail, I need to review this paper again.
Q. Sure.
A. Okay.
Q. Right up above that paragraph.
Do you see where it says, "Several more misclassified children were incorrectly" --
A. Sorry. I have to go back to the methods. This is -- you know, you're asking me to comment on --
MS. BROWN: Take as long as

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you need.
BY MR. WATTS:
Q. I'm really not asking you to comment. I'm asking you, can you see the words on the paper?
MS. BROWN: Yeah, but in order to answer your question, he needs to look --
MR. WATTS: He needs to look at the paper and tell me whether it's there. We don't need to waste ten minutes reading all these articles.
MS. BROWN: But -- well, I just don't think that's fair, sir. If he wanted to comment -- hang on --
MR. WATTS: I'm not asking for a comment --
MS. BROWN: Hold on. Let me put my objection on the record.
MR. WATTS: Okay.
MS. BROWN: The witness has been given a document. He's asked

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for a moment to review and refresh on the document to accurately and truthfully provide testimony in this deposition.
So I'm going to object to anything that doesn't allow him enough time to do that.
MR. WATTS: Okay. Your objection is noted.
BY MR. WATTS:
Q. And my question is, can you see on the screen, "Several more misclassified children were incorrectly transferred from case to non-case status"?
A. I'm able to read those words on the screen. I'm not able to interpret the meaning or the significance of them.
Q. Now, this article that you want to read is one that you cited in your report, right?
A. Correct.
Q. Okay. Now, Avchen was dated what year?

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A. 2011.
Q. Now, with respect to -- go back to 545 for a second.
I want to talk about this expansion of diagnostic criteria.
In your report, you cite to a paper by Wazana. Do you know Dr. Wazana?
A. I do not.
Q. And Wazana, is that the one source she gave with respect to the issue of the expansion of ASD diagnostic criteria, Footnote 27?
A. Sorry, can you repeat the question?
Q. Sure.
Exhibit 403 --
MS. BROWN: Is that your -- I think we're on your report.
MR. WATTS: Yes.
THE WITNESS: The Wazana paper.
Oh, you're talking about my reference in the report to Wazana?

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MS. BROWN: Yep.
THE WITNESS: Oh, okay.
So what was the question?
BY MR. WATTS:
Q. Sure. Paragraph 42.
MS. BROWN: Paragraph 42 of your report.
MR. WATTS: Exhibit 403.
THE WITNESS: That's my report.
MR. WATTS: Page 18, Paragraph 42.
THE WITNESS: 42. Yep.
BY MR. WATTS:
Q. You say, "Additional factors that could drive ASD prevalence rates higher have been described in published literature," and you provide Wazana in 2007; is that right?
A. Yes.
Q. Now, here is my question:
With respect to Wazana, have you read any articles that criticize the work by Wazana as being not legitimate?

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MS. BROWN: Objection. Vague.
THE WITNESS: I may or may not have. I don't recall at this point.
MR. WATTS: Pull up 417, Hertz-Picciotto paper from January of 2009.
Now, is this a study that you told me you had not read before?
A. I believe so.
Q. Okay. Go to Page 8 and 9, and put them up split screen so we can see them both.
MS. BROWN: Okay. Hold on. This is the one we --
THE WITNESS: This is the one that we don't have --
MS. BROWN: It's okay.
MR. WATTS: Okay. Hold on. Hold on. I'll give it to you.
Put up 8 and 9 on the split screen, Erik, please. Thank you.

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And then highlight the bottom of 8, "simulation study by Wazana," all the way to the top of 9.
MS. BROWN: Okay. Hang on one second.
Here is a hardcopy. And, again, if this is something you're not familiar with, take as long as you need to orient yourself to be able to answer the questions.
BY MR. WATTS:
Q. "Simulation study by Wazana et al. suggests that an apparent increase of as much as 28-fold could be explained by a combination of three artifacts: a change in case definition, a decline in age of diagnosis, and better ascertainment. Several problems with this analysis detract from the validity" -- "from its validity and applicability. First, the data they use for the decline in mean age at diagnosis are based on noncomparable cohorts.

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Specifically, they rely on a reported analysis of DDS data used follow-up to a specific calendar date rather than to a specific age. This inflates the decline in age at diagnosis because children from recent birth cohorts are too young for calculation of diagnosed at older age. Using longer follow-up and equivalent follow-up periods, we recalculated the mean age at diagnosis for birth cohorts from 1990 to 1996 to be 5.23, 5.16, 5.12, 5.18, 5.02, 4.90, 4.83, a tenfold smaller shift (0.14 years between 1991 and 1994) than what was assumed in the simulation study of 1.6 years. This shift is evident from our Figure 4.
"Secondly, the extremely large increases found in the simulation observed only in the analysis of cumulative incidence to age four years (labeled 'prevalence' by the authors.) When the simulation's carried out to age 12 years, the magnitude of the explained increase is much less. By this age, the

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impact of age at diagnosis is largely eliminated, and the magnitude of artifactual increases that result from the other two assumptions (change in definition and more efficient) combine to a 2.4-fold increase. This prediction is much smaller than the actual increases in autism rates in the California DDS data, even if we assume, as Wazana et al. did, that all clinicians were using DSM-III in the earlier period (unlikely, given that the DSM-III-Revised had already been adopted) and all clinicians were using the DSM-IV at the end of our study period."
Were you aware of this criticism of Wazana before you cited it?
MS. BROWN: Objection to the form of the question.
THE WITNESS: So Wazana is one reference among many that contributes to my opinions about the increasing prevalence of autism and is very commonly

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accepted in the scientific community.
The fact that there's another paper that criticizes one of the many papers that I cite doesn't change my opinion.
MR. WATTS: Objection. Nonresponsive.
BY MR. WATTS:
Q. Were you aware of this criticism of Wazana when you cited it?
A. So you've already asked me that question.
Q. You didn't answer it.
A. I did.
MS. BROWN: Well, let him answer again.
THE WITNESS: You've asked me if I have seen this paper.
BY MR. WATTS:
Q. Were you aware of it?
A. I -- I had not seen this paper, no.
Q. Okay. Let's go to Volkmar.

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MS. BROWN: Were you done with your answer, Doctor?
THE WITNESS: Only to clarify that in order for me to be thoughtful and careful about my answer as it relates to this paper and this criticism, I need to spend time reviewing it because I haven't seen it before.
BY MR. WATTS:
Q. Okay. Let's go to Volkmar --
MS. BROWN: He's done though, Mr. Watts. Let him finish.
Was there anything else?
THE WITNESS: I'm done.
MS. BROWN: Okay.
BY MR. WATTS:
Q. Let's go to Volkmar. Is that the next study that you cite in Paragraph 43?
Exhibit 403, Paragraph 43.
MS. BROWN: I'll take this.

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Go to your report. We're back to your report.
THE WITNESS: Yeah.
MS. BROWN: He wants to ask you about another citation at Paragraph 43.
Do you need it back?
MR. WATTS: I'll get it back.
THE WITNESS: Yes.
BY MR. WATTS:
Q. So you cite Volkmar in 1988 as examining "a sample of 52 individuals with autism diagnosed by clinical experts and 62 individuals with developmental disability with autism -- without autism and applied criteria from two different editions of DSM.
"Applying the DSM-III criteria to this cohort correctly diagnosed 42/52, or 81 percent, with autism and incorrectly diagnosed 4/62, or 6.5, developmentally disabled individuals as having autism"; is that right?

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A. Yes.
Q. All right. So the net effect is 6 out of 62 were undercounted, 15 percent undercount, right?
A. The purpose of this study was to show that by applying different criteria, you can change the prevalence and the autism diagnosis. And that's what they did.
Q. What was my question?
Was the net effect that the undercount was 15 percent, or 6 of 62?
A. In this small cohort, that shows sort of a proof of concept, yes.
Q. What was the increase in ASD prevalence between 1987 and 1994 when the DSM-III-Revised was in place before DSM-IV came out?
A. I'm not aware of exactly what the increase was between those two years.
Q. Did you provide any studies showing the numerical impact of DSM-III-Revised on subsequent prevalence

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rates?
A. From DSM-III to DSM-III-R? In my report?
Q. Yep.
A. No. This -- this article and the idea behind this section of the report reflects the fact that when diagnostic criteria are broadened, more people are included in the diagnosis. That is inarguable.
Q. Did you provide any studies showing the numerical impact of DSM-III-Revised on subsequent prevalence rates? Yes or no?
MS. BROWN: Objection. Asked and answered.
THE WITNESS: As I said, the premise here is that prevalence has increased. It's increased for a variety of reasons. Here is one example of a reason. Here is one study to support that.
BY MR. WATTS:
Q. Did you provide any studies

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showing the numerical impact of DSM-III-Revised on subsequent prevalence rates?
MS. BROWN: I object. He's answered it three times now. I'm looking at the answer.
BY MR. WATTS:
Q. It's a yes-or-no.
MS. BROWN: It is -- he already answered it. It's there.
BY MR. WATTS:
Q. What studies did you provide showing the numerical impact of DSM-III-Revised?
A. So the study that I am citing here shows the concept that when you change criteria, you can actually have an increase in prevalence, even within the same cohort.
Q. What percentage of the increased prevalence between 1987 and 1994 did scientific studies attribute to the revised criteria in DSM-III-Revised?
A. I have not investigated that

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specific issue.
Q. Okay. Now, with respect to your statement on Page 19, Paragraph 44, you say, "It's noteworthy that the DSM criteria for autism spectrum disorder have continued to expand since autism first became a diagnostic entity in DSM-III in 1980."
Do you see that, sir?
A. Yes.
Q. Are you suggesting that the rate of autism prevalence that we see in this country from 2013 to 2023 is a result of DSM-V diagnostic criteria replacing DSM-IV?
A. I'm suggesting that there's a whole number of factors that contribute to the increase in prevalence and, among them, the change in criteria may contribute, yes.
Q. What percentage of the increase in the rate of prevalence of ASD diagnosis do the studies show is attributable to DSM-V being utilized as

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opposed to DSM-IV?
A. I have not investigated that specific issue.
Q. Well, you cited to Tidmarsh, right?
A. That's a citation, yes.
Q. And Tidmarsh didn't try to quantify that increase of rate, right?
A. Well, the Tidmarsh paper was written -- or was a chapter in 2003.
Q. Okay. So that doesn't have anything to do with DSM-V replacing DSM-IV, agreed?
A. Again, I think we're kind of missing the forest for the trees here, where diagnostic criteria have changed steadily since the autism was first described. That broadening of diagnostic criteria has led to an increase in prevalence, among many other factors.
MR. WATTS: Objection. Nonresponsive.
BY MR. WATTS:
Q. Tidmarsh didn't have

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anything to do with explaining the increase in rate of prevalence of autism spectrum disorder since the publication of DSM-V in 2013, agreed?

A. Tidmarsh was published in 2003. So the answer is no, because the criteria would change in 2013.

Q. Let go to LaSalle in 2023. Exhibit 511, please.

(Document marked for identification as Exhibit Kolevzon 511.)

BY MR. WATTS:

Q. And as we look at the introduction, second sentence, "The prevalence of ASD has been steadily increasing over the past 20 years, from U.S. child estimates of 0.66 percent in 2002, 1.13 percent in 2008, 1.85 in 2016, and 2.27 percent in 2018. Changes over this period in the rate of ASD is in part due to increased awareness and changing diagnoses. However, even estimates that account for diagnostic changes still

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leave an apparent increase that cannot likely be explained by genetics alone."

Is that what it says?

A. Those are the words on the page.

Q. And at the bottom of that first paragraph, it says, "Together, these findings have demonstrated that ASD etiology is decidedly complex, involving hundreds of genes and interactions with environmental factors."

Is that what it says?

A. So, again, those are words on the page. This is a paper that I have not reviewed.

But I need to clarify some points of this before I agree with it in totality.

Q. Now, the DSM-V was published in May of 2013, right?

A. I don't recall the exact month, but it was definitely 2013.

Q. Okay. If your report says that, would you buy what you said?

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A. Yes.

Q. Okay. Have you read any of the studies that have calculate the percentage of change of prevalence rates in autism spectrum disorder that had been attributed to DSM-V over DSM-IV?

MS. BROWN: Objection. Lacks foundation.

You can answer.

THE WITNESS: So I've read some of the studies, but I don't have an immediate memory of them at this moment.

BY MR. WATTS:

Q. Let me take you through some of them. And I want to start with just a reference document. And this is Exhibit 481.

(Document marked for identification as Exhibit Kolevzon 481.)

BY MR. WATTS:

Q. It's a young man named Toby Rogers, thesis in some school in

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Australia.

And Page 19. I'm not going to ask you anything about the thesis, just what he's citing.

MR. WATTS: Page 19, Erik.

MS. BROWN: I'm going to object to this gentleman's thesis as lacking foundation.

And just for the record, I have like a three-inch stack of printed dissertation here that I am aware that Dr. Kolevzon has never seen before.

So lacks foundation. And to the extent you need time to at least flip through Mr. Toby Rogers' paper.

BY MR. WATTS:

Q. Go to Page 19. I'm not going to ask you about anything else.

Here is my question:

On Page 19 --

MR. WATTS: Blow up Barton, Robins -- just that paragraph,

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Erik. Yep.
BY MR. WATTS:
Q. And he cites to Barton, Robins, Brennan, and Fein in 2013, and Mazefsky, McPartland, Gastgeb and Minshew in 2013, who "concluded that the more precise definition of autism in DSM-V will result in fewer numbers of children diagnosed with autism."
Do you see that?
A. I see the words that are written on the page.
Q. Are you familiar with the Barton paper in 2013 or the Mazefsky paper in 2013?
A. I'm familiar with the concerns at the time that the change in diagnostic criteria would sort of lead to people losing their diagnosis.
MR. WATTS: Objection --
THE WITNESS: I'm not familiar with this specific paper. Or if I've read it, I don't recall.

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BY MR. WATTS:
Q. Okay. How about the Maenner paper in 2014, have you read it?
A. Maenner is one of the main authors on the CDC papers. So I don't know this exact reference that they are referring to. But I may or may not have read it.
Q. And it says that the DSM-V definition would lead to an 11.5 percent decrease in the number of children diagnosed with autism.
Did you know that?
A. You're reading from a person I never heard of, for a dissertation that is not peer-reviewed, and for a reference that I had no knowledge of --
Q. Let's go to Maenner's paper --
MS. BROWN: Wait. Let him finish, though, please.
BY MR. WATTS:
Q. Let's go to Maenner's paper that was peer-reviewed, Exhibit 433. I

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think that's --
(Document marked for identification as Exhibit Kolevzon 433.)
MS. BROWN: And for the record, where did Tony Rogers' [sic] dissertation come from? How can we --
MR. WATTS: In Australia. And I only used it for purposes of pointing out two studies to see whether he knew it was there. I'm not going to ask any more --
MS. BROWN: I understand. I just want, for the record, how would one access this paper.
MR. WATTS: I can -- well, Number 1, you've got it. And Number 2, I'll show you how.
MS. BROWN: Okay. Let's just add to the record, before we close the deposition, a link, a cite, something that would allow someone in the public to access

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this.
MR. WATTS: Sure.
And I've already given you a disc with the documents, so you'll be able to see it there as well.
MS. BROWN: I know. It's just we don't have time to review the three inches of his dissertation. So I want to know how to access it.
MR. WATTS: So what you do is you type in "Toby Rogers" or the title and you turn on the Google machine and it will come up.
MS. BROWN: Okay. That's what I wanted to know.
BY MR. WATTS:
Q. Okay. Now, enough of this. Exhibit 433. Matthew Maenner.
Have you read this -- have you read this article before?
MS. BROWN: Hang on.

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BY MR. WATTS:
Q. Have you ever seen it before?
A. I recall this paper coming out. I don't remember the details of it. I'd have to review it if you wanted me to be --
Q. So --
MS. BROWN: Wait. Let him finish.
BY MR. WATTS:
Q. Maenner's article is entitled, "Potential Impact of DSM-V Criteria on Autism Spectrum Disorder Prevalence Estimates."
Published in JAMA Psychiatry in March of 2014. And your testimony is you recall it coming out but you don't recall reading it, correct?
A. No.
MS. BROWN: Well --
THE WITNESS: My testimony is that I recall it coming out, but I don't remember the details

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off the top of my head.
BY MR. WATTS:
Q. Okay. So --
A. And I'll also say that Maenner is using methods from the CDC-monitoring sites, which is one of the methods that has contributed to increasing prevalence.
Q. And Maenner, on Page 2 of 20, in the results, "Based on these findings, ASD prevalence per 1,000 for 2008 would have been 10.0 (95 percent confidence interval, 9.6 to 10.3) using DSM-V criteria compared with reported prevalence based on DSM-IV-TR criteria of 11.0 to 11.7."
Did I read that correctly?
MS. BROWN: I object as lacking foundation.
This is an article he said he's not familiar with.
THE WITNESS: Yes. But --
BY MR. WATTS:
Q. Okay.

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A. Hold on. Hold on. I'm sorry.
We have to go, just for clarification, to the methods. All right. So the methods used for this study were based on coded behaviors documented in children's medical records and educational evaluations. These are not proxies for the diagnosis. This is the problem with the CDC methods overall. And so the fact that Dr. Maenner has now taken the CDC methods and applied it to the DSM-III -- DSM-IV and the DSM-V doesn't mean that I necessarily would agree with these results.
Q. Did you put this in your materials considered?
A. Did I put what in my materials considered?
Q. The Maenner paper.
A. This particular paper? I don't know if I did or not.
Q. Okay. Let's go to the Sturmey paper of 2013, Exhibit 437.

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(Document marked for identification as Exhibit Kolevzon 437.)
BY MR. WATTS:
Q. The abstract --
MS. BROWN: Hang on. Let's just give him a second to get the copy so he can answer the question.
MR. WATTS: Pull up the abstract.
BY MR. WATTS:
Q. "Systematic review of empirical papers comparing the application of DSM-IV and DSM-V diagnostic criteria for Autism Spectrum Disorders identified 12 papers. The application of DSM-V diagnostic criteria resulted in approximately a one-third reduction in Autism Spectrum Disorders."
Did I read that correctly?
A. Those are the words on the page, yes.
Q. Did you cite to any of those

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12 papers saying that DSM-V resulted in a contraction of the number of autism spectrum disorders being reported?
A. I don't know whether those papers are cited in my reference list or not.
Q. Okay. In terms of the quantum of that subtraction -- go to Page 251.
"In reviewing those 12 papers, the median overall change in diagnosis of ASD from all papers was negative 36.97 percent."
Do you see that, sir?
A. I see what they've said on the paper.
Q. Now --
A. This is not in -- not consistent with the way that it's viewed in the community. It's not consistent with my experience as a clinician.
The idea that we're debating whether or not change in diagnostic criteria have led to an increase in

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prevalence to me is misguided.
MR. WATTS: Doctor, nonresponsive.
MS. BROWN: Object.
BY MR. WATTS:
Q. My question is, did you ever see Sturmey's paper before I just showed it to you?
A. I don't recall whether I have or haven't.
Q. Let's go to Zander's paper in 2015, Exhibit 443.
(Document marked for identification as Exhibit Kolevzon 443.)
BY MR. WATTS:
Q. A "New DSM-V Impairment Criterion: A Challenge to Early Autism Spectrum Disorder Diagnosis?" by Eric Zander, published online in June 28, 2015.
Did you cite this in your materials considered?
A. I don't know if I did or

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didn't.
Q. On the second page -- I'm sorry, on Page 3640.
MS. BROWN: Let's give him a minute to familiarize himself with the paper.
BY MR. WATTS:
Q. Well, as we're familiarizing ourself.
MR. WATTS: Let's go back to 3636. I want to set the stage.
MS. BROWN: I'm just going to object. The hardcopy is missing pages, and to the extent that he's not familiar with it, we need to get him a copy that he can read to answer your questions.
MR. WATTS: 3636.
THE WITNESS: I'm sorry, I'm still on this paper.
MS. BROWN: Take your time. Take you time.
This -- my copy was missing pages. If you want a complete

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one, you might want to look at this.
MR. WATTS: Erik, did you pre-highlight this stuff?
TRIAL TECH: Yes.
MR. WATTS: Can you push click so the highlighting comes up so we can figure it out?
TRIAL TECH: On Page 3636?
MR. WATTS: Yeah.
BY MR. WATTS:
Q. "The objective of the current study was to investigate the impact of the new DSM-V impairment criterion on diagnosing ASD in toddlers and young preschoolers."
A. It looks like -- and I'm just seeing this for the first time. It's focused on the impairment criterion using an adaptive behavior scale.
Q. Now, when you say you're seeing it for the first time, you hadn't seen it before I just showed it to you?
A. I don't recall seeing this

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before.
Q. Okay. If we go to 3641. The conclusion after that objective was set says, "Our findings indicate that a strict application of this DSM-V criterion would compromise the possibility for very young children to get an ASD diagnosis despite exhibiting the defining symptomatology."
Do you see that, sir?
MS. BROWN: I object to lacking foundation. He said he hasn't seen the article before.
BY MR. WATTS:
Q. You hadn't seen it, had you?
MS. BROWN: Well, he asked for a minute to read --
THE WITNESS: You asked me two different questions.
MS. BROWN: -- and familiarize himself with the article.
THE WITNESS: You asked me two questions. One, do I see the

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words on the page? The answer's yes.
Two, if I see -- I don't recall seeing this paper.
BY MR. WATTS:
Q. Let's go to Bennett, 2016, Exhibit 447.
(Document marked for identification as Exhibit Kolevzon 447.)
BY MR. WATTS:
Q. "A Meta-Analysis of DSM-V Autism Diagnoses in Relation to DSM-IV and DSM-IV-TR?"
Published in the Review Journal of Autism and Developmental Disorders in 2016. The author is Matthew Bennett.
MS. BROWN: And, again, Doctor, if you haven't seen this before, take as long as you need to familiarize yourself with it.
BY MR. WATTS:
Q. Page 2, it says, "All 24

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studies identified indicated a reduction in the number of people with a DSM-IV or DSM-IV-TR ASD diagnosis being eligible for a DSM-V ASD diagnosis" -- and "a 35 and 37 percent reduction respectfully."
MS. BROWN: And I object as lacking foundation.
Mr. Watts, what we're doing is just reading random sentences in articles that he's testified he would like a minute to review and he has not seen before. So I object --
MR. WATTS: Well, that's my question, have you see it before.
MS. BROWN: Wait. Let me just put this on the record.
MR. WATTS: And, Alli, your job is to say "Objection to form."
MS. BROWN: I understand. But what you're doing is not fair.
MR. WATTS: I know, but when you're giving speeches, you're coaching the witness.

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MS. BROWN: Well, first of all, that's not -- that's not fair.
MR. WATTS: You can't give a speech every time. You can say, "Objection. Form."
MS. BROWN: I need to preserve an objection to this line --
MR. WATTS: Have you read the deposition protocol order?
MS. BROWN: I have.
MR. WATTS: Okay. So --
MS. BROWN: And I am absolutely complying it -- complying with it.
MR. WATTS: Okay.
MS. BROWN: But what doesn't comply with it is when a witness asks for time to look at something and you continue to just read random sentences.
I object to that. And it lacks foundation. And it's

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improper.
BY MR. WATTS:
Q. Doctor, have you ever seen this paper before?
A. I can't say whether I have or I haven't.
MR. WATTS: Go to Baio, 2018, Exhibit 475.
(Document marked for identification as Exhibit Kolevzon 475.)
MS. BROWN: Please finish your answer.
Did you have something you were answering?
THE WITNESS: I feel like it's important to understand all of these papers in their context, right.
When the DSM-V came out there were concerns that people would lose their diagnosis, and so lots of papers came out on both sides of this issue, some showing

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they wouldn't, some showing that they would, and it was a very kind of heightened time.
Since then, it's clear that prevalence rates have increased, and it's clear that many, many, many factors have contributed, including the broadening diagnostic criteria.
BY MR. WATTS:
Q. You done?
A. Yes.
MR. WATTS: Objection. Nonresponsive.
MS. BROWN: Objection.
BY MR. WATTS:
Q. Go to Exhibit 475.
Have you seen the Baio paper, published by the Centers for Disease Control and Prevention in 2018?
MS. BROWN: Give us a minute to just get the hardcopy in front of him. Take a minute.
BY MR. WATTS:

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Q. Doctor, my only question is, have you seen this before?
A. Yes.
Q. Okay. Let's go to Cartolano in 2018, Exhibit 476.
(Document marked for identification as Exhibit Kolevzon 476.)
MS. BROWN: One second.
BY MR. WATTS:
Q. And in fairness to you, I'm sure, I won't blame you if you haven't seen this one. It looks like it's some sort of a graduate student study.
"Under the Umbrella: Redefining the Spectrum of Autism."
Without being critical, have you ever seen this before?
A. Definitely not.
Q. Okay. Let's keep going.
You know who Dr. Fombonne is?
A. Yes.
Q. Exhibit 477.

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(Document marked for identification as Exhibit Kolevzon 477.)
BY MR. WATTS:
Q. You cited this paper in your reliance materials at Footnote 25.
And the question I have is on Page 9 of 13, Footnote 1, where Dr. Fombonne --
A. Hold on.
MS. BROWN: Just one second, please.
BY MR. WATTS:
Q. Page 9 of 13.
A. Just give me a second.
Q. Yeah.
In Footnote 1 it says, "Calculated by the author from Baio et al. (2018), Page 12."
Then it says, The DSM-IV-TR prevalence in the same subsample was 1.77 percent (4,236 plus 4,222 over 20 -- 263,775). The DSM prevalence is reduced by 18.1 percent compared to that of the

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DSM-IV-TR.

Have you ever talked to Dr. Fombonne about his calculation about the reduction in prevalence under DSM-V versus DSM-IV?

MS. BROWN: Objection. Lacks foundation.

THE WITNESS: I've talked to Dr. Fombonne about the rise in prevalence of autism, but not about this specific calculation.

BY MR. WATTS:

Q. Okay. Let's go to Exhibit 490.

(Document marked for identification as Exhibit Kolevzon 490.)

MR. WATTS: Kalra in 2021.

MS. BROWN: What's the number? 490?

BY MR. WATTS:

Q. "Comparison of diagnostic criteria for autism spectrum disorder (ASD) using Diagnostic and Statistical

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Manual (DSM) and International Classification of Diseases (ICD)."

In fairness, this is a study in Northern India. Have you ever seen this?

A. I don't recall if I have or not.

Q. Okay.

MS. BROWN: Well, if you have not -- I mean, he should probably take a minute to check if you want a truthful answer.

MR. WATTS: I'm happy with the answer he gave, because I wouldn't expect anybody to see it anyway.

BY MR. WATTS:

Q. We'll go to one he should have seen.

Exhibit 498, the CDC, in 2022.

(Document marked for identification as Exhibit Kolevzon 498.)

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BY MR. WATTS:

Q. I assume you have seen this.

MS. BROWN: Take a minute to answer.

THE WITNESS: This exhibit is a?

BY MR. WATTS:

Q. Press release.

A. Press release from the CDC.

Q. Okay. And --

MS. BROWN: Let's just give him a minute to read it.

BY MR. WATTS:

Q. It says, under "Main findings," "The findings suggested that estimates of the number of children with ASD might be lower using the current DSM-V criteria than using the previous DSM-IV-TR criteria."

Do you see that, sir?

MS. BROWN: Object as lacking foundation.

If you need a minute to look at this and understand what this

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press release is about, please take it.

THE WITNESS: So I see the words on the page, but I don't know on what basis they are making this conclusion, and I don't know what study they are relying on.

BY MR. WATTS:

Q. Did you cite to this press release?

A. I don't know if I cited to this press release or not.

Q. Have you ever seen that the CDC says that the number of children with ASD might be lower using the current DSM-V criteria rather than using the DSM-IV-TR criteria?

MS. BROWN: I object. That misstates this document and the CDC's position.

THE WITNESS: I don't know what this reference refers to specifically in terms of studies.

I know that the CDC methods

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of calculating prevalence of autism are fundamentally flawed.

BY MR. WATTS:

Q. Have you called the CDC and told them that?

A. No.

Q. Have you written to Congress telling them that their money is being spent on a fundamentally flawed calculation at the CDC?

MS. BROWN: I object. I object. That lacks foundation --

THE WITNESS: I don't see that as my role.

BY MR. WATTS:

Q. Have you published a single article stating that the CDC prevalence calculation methodology is mistaken?

MS. BROWN: Objection to the form of the question.

THE WITNESS: So the scientific community that examines these issues has raised concerns about the methods, about how these

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methods are contributing to estimates that are overblown.

MR. WATTS: Objection. Nonresponsive.

MS. BROWN: Object.

BY MR. WATTS:

Q. Has Alexander Kolevzon published a single word saying that the CDC calculations of autism spectrum disorder prevalence rates is in error?

MS. BROWN: Objection. Lacks foundation.

THE WITNESS: You need to be specific about when you say about -- when you say published.

BY MR. WATTS:

Q. Like written an article telling the scientific community the CDC has got it all wrong.

MS. BROWN: Objection.

BY MR. WATTS:

Q. Have you done that?

A. I have definitely spoke about my concerns with the methods, both

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professionally and in my teaching. And my view is consistent with the general view.

And, by the way, it's just factually correct. You cannot make the diagnosis of autism based purely on school records and symptoms that appear in school records that are consistent with the criteria.

MR. WATTS: Objection. Nonresponsive.

MS. BROWN: Object.

BY MR. WATTS:

Q. Have you published anything saying the CDC calculation rate is in error?

MS. BROWN: Objection. Asked and answered.

You can answer again, Dr. Kolevzon.

THE WITNESS: As I said, when I teach, when I speak to professionals, when I give presentations, and this issue

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comes up, I'm certainly most vocal about it.

BY MR. WATTS:

Q. Is there a reported publication?

A. Is there a peer-reviewed reported publication?

Q. Yes, sir.

A. I don't recall.

Q. Okay. Let's go to the younger age of diagnosis.

Now, you reference that in 2007 the American Academy of Pediatrics published a policy recommending ASD-specific screening at 18 to 24 months, right?

A. Yes.

Q. And to the extent that that caused a bump in prevalence rate, would you give me that that's a onetime bump?

MS. BROWN: Objection to the form.

THE WITNESS: No.

BY MR. WATTS:

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Q. Okay. So from the standpoint of what happened in 2007, they recommended screening at 18 months, right?
A. 18 and 24 months.
Q. Yes.
And has there been any change in the date of the recommendation for screening in the last 16 years since 2007?
A. I'm not sure I understand the question.
Q. Sure.
Has it changed, since that 2007 recommendation, to start diagnosing between 18 and 24 months?
MS. BROWN: Objection. Vague.
You can answer if you understand.
THE WITNESS: Has the recommendation to screen for autism at 18 to 24 months changed since 2007?

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BY MR. WATTS:
Q. Yeah. They haven't brought it down to six to eight months, is my point.
A. No.
Q. Okay.
A. But the compliance with that recommendation has increased steadily.
Q. Now, you cite in support of this argument to a paper called Sheldrick on Page 20, Footnote 35.
And you say that Sheldrick shows "that ASD screening and early intervention of less than 3 years old is associated with a 60 percent increase in ASD diagnosis"; is that right?
A. I'd have to pull up this paper to look at it more carefully. But that's what I've written on the page, yes.
Q. Now, you realize Sheldrick was simply saying we needed to go into communities of color that are not being screened and start screening, right?

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MS. BROWN: Objection. Lacks foundation.
THE WITNESS: The point is that if you don't screen, you don't detect cases; and when you do screen, you do detect cases.
BY MR. WATTS:
Q. Okay.
A. And so when screening became mandated, more cases were detected.
Q. Okay. And in Sheldrick they said, look, the services failed to provide ASD screening to Spanish-speaking families, and so we need to screen for Spanish-speaking families, right?
MS. BROWN: If we want to talk about this paper, can we pull it up.
MR. WATTS: No.
BY MR. WATTS:
Q. Is that what happened? You cited it, you should know it.
MS. BROWN: Well, how's he going to know all these papers --

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MR. WATTS: "Objection. Form," Alli. Cut it out.
MS. BROWN: I object to the form. I object to the form.
But let's be fair with him. You want a truthful answer --
BY MR. WATTS:
Q. Is that -- do you know what Sheldrick did or not?
A. As I understand it, this is not an exercise in my memory. I did review this paper carefully, and I'm happy to go back and review it again.
Q. Do you know that it had to do with going into communities with Spanish-speaking-only parents?
A. Again, I'm happy to review the methods again right now.
Q. Now, let me take you back to Exhibit 417, which is the Hertz-Picciotto "Rise in Autism, Role of Age At Diagnosis." This is the one you told me you hadn't seen, right?
A. I have not seen this paper.

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Q. Have you looked at any of Dr. Hertz-Picciotto's work with respect to the rise in autism prevalence in California?

A. I'm sure I have. I don't recall exactly what all the findings were at this moment.

Q. And you realize that she says, is the changing age of diagnosis can only explain a 12 percent increase?

A. I don't know what Dr. Hertz-Picciotto says or doesn't say specifically.

Q. Go to Page 3 of 20. Let's just pull it out there.

MS. BROWN: Hang on. Let's just get the hardcopy in front of him.

This is something we've already marked?

THE WITNESS: This is the one that -- this is not the full version of it, though.

MS. BROWN: I think this is

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it, right?

So, Counsel, this is the one that I think we had borrowed your copy for because we only had the abstract. And we need to borrow it back, please.

MR. WATTS: I'm giving it back to you.

MS. BROWN: Thank you.

BY MR. WATTS:

Q. The last sentence of the results on Page 3 of 20 says, "Changing age of diagnosis can explain a 12 percent increase," right?

MS. BROWN: Hang on. Object as lacking foundation.

Let's let him get to the hardcopy and review what he needs to, to answer your question.

BY MR. WATTS:

Q. Right there.

A. That's what it says, yes.

Q. Now, if changing age at diagnosis explains a 12 percent increase,

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and there's a 685 percent increase, that means that of the increase, only 1.75 percent of it is explained by changing age of diagnosis, doesn't it?

MS. BROWN: Objection. Assumes facts. Lacks foundation.

THE WITNESS: So the conclusion that's written here is that the changing age at diagnosis can explain a 12 percent increase, and part of that includes also milder cases.

BY MR. WATTS:

Q. So that's 56 percent. So you add those together and that's 68 percent, but we had 685 percent increase.

A. So there are many, many factors that contribute to the increase in prevalence.

The most likely of which and the most, kind of, profound of which is misidentification of cases.

Q. Doctor, you listed five

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factors, so I'm asking about them, and the changing age of diagnosis, which is Category C, the one study that I've been able to pull up says it increases it by 12 out of 685 percent.

Do you have another study you can point me to?

MS. BROWN: Objection to the form of the question.

THE WITNESS: So at this moment, off the top of my head, I do not.

But when you take a swath of people at a certain age, like eight, and you look for people that have been diagnosed with autism, if they were previously diagnosed at six or seven and now they are being diagnosed at two or three, you are going to include more people in your study.

BY MR. WATTS:

Q. Sure. But they are not going to be rediagnosed when they turn

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six or seven, right?
A. No, they would -- they would count as a case.
Q. So when I said it was a onetime phenomenon, that's what I meant.
They are counted. They are in the system. They are not counted again when they turn six or seven again, right?
A. Culturally, what this mandate did was it sensitized pediatricians across the country to the importance of screening for autism, and so whether they did it properly at 18 to 24, or they were desensitized to it and did it at six or seven, the average age of diagnosis went down. It just did.
Q. Doctor, and it explained a 12 percent increase, but we've got a 700 percent increase.
A. The point being --
Q. Do you have another study that says the changing age of diagnosis had a statistically more impactful result

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in explaining the overall increase of prevalence?
MS. BROWN: Objection. Compound. Misstates the study. Misstates the evidence.
BY MR. WATTS:
Q. Do you have a single other study?
A. You're pointing to a single study.
Q. Do you have one?
A. I don't have one off the top of my head right now.
Q. Okay. Let's go to the changes in law in 1991.
And I want to go to your report, Exhibit 403. Page 20, paragraph 46, please.
All right. Now, Paragraph 46 and 47, you reference something that happened in 1991 called the Individuals with Disability Education Act?
A. And it continues to be

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relevant today.
Q. And you cite Croen in 2002; is that right?
A. That's one of the references that supports that, yes.
Q. Well, help me with any other reference that supports that, that you bothered to cite in this -- this report?
MS. BROWN: Did you say that you bothered to say?
MR. WATTS: I did.
MS. BROWN: I object. That's argumentative.
BY MR. WATTS:
Q. Okay. So there's other ones, but you didn't give them to me, so let's talk about the one you did give. And that's Croen, right?
A. Yeah. And I can also rely on my 20 years of experience in seeing thousands and thousands of kids being misdiagnosed for the purposes of educational services.
Q. Doctor, did you give me a

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single other citation other than Croen in 2002?
MS. BROWN: Objection to the form of the question. Misstates the report.
THE WITNESS: As one reference to support this idea that's commonly accepted in the scientific community as truthful, I've cited one reference.
BY MR. WATTS:
Q. It's the only reference you gave me, right?
MS. BROWN: Objection.
THE WITNESS: In addition to my 20 years of experience.
BY MR. WATTS:
Q. And so let's split this up.
Did I only get one reference, Croen of 2002, yes or no?
A. I provided one reference to support this statement.
Q. Okay. And you say that reference is so excellent, it's commonly

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accepted as proving your point, right?
A. That mischaracterizes what I said.
What I said is that it's commonly accepted in the scientific community, based on decades of clinical experience, that when this law changed, families decided they wanted services and they got the -- the autism diagnosis in order to get services; it became prescriptive.
Q. And as your silver bullet, you give me crown -- Croen of 2002. That's it?
A. It's not a --
MS. BROWN: Objection. Argumentive.
Go ahead.
THE WITNESS: It's not a silver bullet. It is a reference that supports the idea.
BY MR. WATTS:
Q. Do you have a single other reference you want to share with this

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jury other than Croen 2002?
MS. BROWN: Objection. Other than what he already testified to.
THE WITNESS: I think I -- as I've said, at this time, sitting here today, I do not have another reference. That isn't to say that another reference doesn't exist.
BY MR. WATTS:
Q. Now let's visit about Croen in 2002.
MS. BROWN: Were you done?
THE WITNESS: Sure.
BY MR. WATTS:
Q. Croen in 2002, you dropped Footnote 36.
MR. WATTS: Can we show -- just pull up 47 and go down to the footnote so we can see it.
No. No. Take that off.
Okay. I want only Paragraph 47, and I want to see

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Footnote 36.
MS. BROWN: Yep. Mm-hmm.
And Dr. Kolevzon needs a break as soon as your question is not pending anymore.
MR. WATTS: As soon as we get done with Croen, we'll take a break. Okay?
MS. BROWN: Good.
BY MR. WATTS:
Q. All right. You see how in Footnote 36 you cite to Croen, "Descriptive epidemiology of autism in a California population: Who is at risk?"
Is that right?
A. That looks to be the Croen reference, yes.
Q. Okay. And then your only other reference is -- go to 37 and 38 -- is Id. at 214; is that right?
MR. WATTS: Show -- show 36, 37 and 38.
BY MR. WATTS:
Q. So you've given three

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footnotes, 36, 37, and 38.
Id. means same paper, right?
A. Yeah.
Q. Now, I'm curious, Footnote 36 has a paper range of 217 to 224, and your Id. is the 214, which is outside that range.
That's a mistake, isn't it?
A. Go back. Sorry.
Q. Do you see 36?
A. Yep.
Q. And the paper range is 217 to 224, right?
A. Yes.
Q. So it can't be an Id. at 214. It's got to be something went wrong here, right?
A. Most likely.
Q. Okay. Now, let's talk about what went wrong.
MS. BROWN: Can we give Dr. Kolevzon a bathroom break first, though?
MR. WATTS: Sure.

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MS. BROWN: Okay. Perfect.
THE VIDEOGRAPHER: The time right now is 11:02 a.m. We are off the record.
(Short break.)
THE VIDEOGRAPHER: The time right now is 11:10 a.m. We're back on the record.
BY MR. WATTS:
Q. Okay. Doctor, we've got Footnote 36 which cites to "Descriptive epidemiology in a California population: Who is at risk?"
Journal of Autism and Developmental Disorders, 2002, Volume 32, Pages 217 to 24.
We clearly have a typo in Footnote 37 and 38, because it doesn't fit within the range.
But let's go to the paper, which is Exhibit 408, "Descriptive Epidemiology."
(Document marked for identification as Exhibit

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Kolevzon 408.)
BY MR. WATTS:
Q. This is the paper that you cite to in Footnote 36, right?
MS. BROWN: Hold on.
Okay. Got it.
BY MR. WATTS:
Q. Is that right, Doctor?
A. Yeah. They seem consistent.
Q. Yeah. And I'm going to clean it up here in a second.
Go to Page 219, because there's one thing in this paper that I want you to see.
It says, "Our series of children with autism is larger than the total number of children included in all previous epidemiologic studies of autism combined."
Do you see that?
A. I see where it says that, yeah.
Q. Okay. Now, I think what happened, and not being pejorative about

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it, is you cited originally to this, and you meant to cite to another one, which I'm going to take you to, which is 409.
So let's just clean this up, if we could.
(Document marked for identification as Exhibit Kolevzon 409.)
BY MR. WATTS:
Q. 409, in fairness to you, is in the same journal. It's entitled, "The Changing Prevalence in Autism in California," and it starts on Page 207 and includes Page 214.
Do you see that?
A. Yeah. In looking at my -- yeah, in looking at my description of the paper, the 36 reference is wrong, and it should be to this.
Q. Right. Okay. So when you cited to 408, you meant to cite to 409. That's where we are, right?
A. Yes.
Q. Okay. Now let's go to 409.

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That's what I thought. And these things happen. It's not a big deal.
A. Yeah.
Q. But let's go to 409.
Okay. On Page 213 --
A. Just let me orient myself one second.
Q. In the second column, it says, "The marked increase in the prevalence of autism and MR observed for children born in 1990-1992 provides support for the hypothesis that improvements in detection may have contributed to our results."
And then go to the next one.
"Changes in diagnostic practices during the study period might also have contributed to the observed increase."
That's what you meant to cite to, right?
A. Yes.
Q. Okay. Now, this is the one

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study, Exhibit 409, that you cited to, by intention anyway, to support your argument in this particular point dealing with the change of special education law, right, Croen in 2002?

A. So it's very clear that the change in special education law led to a shift in diagnostic practices, and this study provides some evidence that, in fact, that's true.

Q. It's the one study you provided to support it, right?

MS. BROWN: Objection. Misstates testimony.

THE WITNESS: I used this study to support that statement.

BY MR. WATTS:

Q. And only this study?

MS. BROWN: Objection. Misstates testimony.

THE WITNESS: This is a study that I've used.

BY MR. WATTS:

Q. Was there any other study

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used?

A. I haven't used any other studies in this particular reference.

Q. All right. Now, the reason I ask that question is, are you familiar with a Dr. Blaxill, B-L-A-X-I-L-L?

A. I am not.

Q. Let's look at what he had to say about Croen in 2002.

Exhibit 410, please.

(Document marked for identification as Exhibit Kolevzon 410.)

BY MR. WATTS:

Q. This is a commentary by Blaxill, Baskin, and Spitzer on Croen, 2002, "The Changing Prevalence of Autism in California."

Have you seen this commentary before?

A. I don't recall if I have or not.

Q. And in the introductions, just get to the meat of it.

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MS. BROWN: Just give him a minute to look at it.

BY MR. WATTS:

Q. It says, "Their calculations purport to demonstrate that over 100 percent of the increase in autism from 1987 to 1994 is an artifact of changes in diagnostic practices. In your editorial commentary, Eric Fombonne praises the study, and claims Croen et al. carefully analyzed the California dataset. We disagree."

Did I read that correctly?

A. It appears that Dr. Blaxill, or Mr. Blaxill, disagrees, yes.

Q. Let's go to his conclusion on Page 226.

"Croen et al. support arguments to set aside the growing body of evidence that we have an epidemic of autistic diseases. They have suggested that 'diagnostic substitution' accounts for an apparent increase in the incidence of autism in California that is not real.

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This hypothesized substitution is not supported by proper and detailed analyses of the California data. On the contrary, California continues to provide the strongest evidence for the explosion in the incidence of autism."

Did I read that correctly?

A. I don't agree with that statement, but you did --

Q. I didn't ask that. Did I read it correctly?

MS. BROWN: Let him finish, please.

THE WITNESS: You read it correctly.

BY MR. WATTS:

Q. Okay. Have you ever seen this before today?

A. I don't recall seeing this, physically.

Q. Now, Croen in 2002 is the one study you provided in support, and yet Blaxill is saying it's wrong, right?

MS. BROWN: Objection.

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Misstates the paper.
THE WITNESS: So, again, I can represent the view of the scientific community in saying that there's clearly a consensus that younger age of diagnosis, change in diagnostic criteria, and educational shifts in sort of regulatory requirements have led to an increase in the diagnosis of autism. And there are some studies that support that, and, I imagine, there are some studies that don't support that.
But, overall, the totality of the evidence suggest that, in fact, that's the case.
MR. WATTS: Objection. Nonresponsive.
MS. BROWN: Objection.
BY MR. WATTS:
Q. The one study that you cited is criticized as being wrong by Blaxill, which you have never seen before today, right?

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A. I don't have the opportunity --
MS. BROWN: Objection to form.
Go ahead.
THE WITNESS: I don't have the opportunity to evaluate Blaxill's commentary.
It's a commentary. It's not even peer-reviewed. I don't think it's fair for me to comment on it.
BY MR. WATTS:
Q. Okay. Well, I'll tell you what. Let's comment on Exhibit 411, which is what Croen said about her own study after reading Blaxill.
Exhibit 411 is entitled "Response: A Response to Blaxill, Baskin, and Spitzer on Croen (2002), 'The Changing Prevalence in Autism in California,'" by Croen and Grether.
(Document marked for identification as Exhibit

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Kolevzon 411.)
BY MR. WATTS:
Q. Have you ever seen Croen's response published in April 2003 about the criticism to her 2002 study that you cited to?
A. I don't recall.
Q. And if we look at --
MR. WATTS: Let's go to Column 2, Eric. First page.
BY MR. WATTS:
Q. Says, "Blaxill et al. correctly point out that age patterns of enrollment in this service system may be substantially different for autism and idiopathic MR and that truncated follow-up for children born during the more recent study years might differentially affect the observed trends in prevalence of these two disorders over the study period. That is, not only did we underascertain autism in the later years (which we acknowledged), but we may have underascertained MR to a

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substantially greater degree (which we did not acknowledge). Blaxill et al. assert that the observed decline in idiopathic MR could be an artifact of a relatively later average age of entry into the system for children with this disorder."
Did I read that correctly?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: It's impossible for me to comment on this without reading it.
BY MR. WATTS:
Q. Did I read it correctly?
A. You read the words on the page.
Q. Okay.
A. But I can't make any kind of opinion about this.
Q. Because you've never seen it before?
A. If I have seen it before, I don't remember.

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Q. You did not cite to it, did you?
A. It was not in my citations.
Q. Let's go down further on the column.
"As a way of addressing the criticisms by Blaxill et al. with our original dataset, we have conducted a reanalysis limiting the data to children who had the same number of years of follow-up across all study years."
And if we go to the next page, they conclude, based on that reanalysis, that, "diagnostic substitution does not appear to account for the increased trend in autism prevalence we observed in our original analysis."
Did you know that they had to reanalyze and retract what they said before?
MS. BROWN: I object to the reading of part of that sentence as incomplete.

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THE WITNESS: There's no doubt that people who were previously diagnosed with intellectual disability are now being diagnosed with autism spectrum disorder.
There's no doubt that prior to 1990 people with autism were not guaranteed educational services, whereas afterwards they were.
And there's no doubt that that's some diagnostic substitution going on.
MR. WATTS: Objection. Nonresponsive.
MS. BROWN: Object.
BY MR. WATTS:
Q. Were you aware that Croen in 2003 had to acknowledge that her methodology that you cited to, she did in 2002, was wrong?
MS. BROWN: I object. That misstates the paper.

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THE WITNESS: In order for me to comment on this paper, I need time to read it, understand the methods, understand exactly what she's saying needed to be changed or not changed. I don't have time to do that.
BY MR. WATTS:
Q. Because you've never seen it before today, have you?
A. If I've seen it, I don't remember. So in order to comment on it, I need to read it carefully.
Q. And let's go to Page 229.
"We agree with Blaxill et al. that the slight degree of diagnostic substitution we observed in these samples would not explain the dramatic increase in the probability of becoming a DDS client for autism by age four."
Did I read that correctly?
A. You read the words on the page.
Nobody is implying that

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diagnostic substitution is the explanation for the entire increase in prevalence. It's one among many, many factors.
Q. And, in fact, you know that this same dataset was later analyzed, and it was only a quarter of the increase, don't you?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: I'm not aware of that specifically.
BY MR. WATTS:
Q. Well, in previous cases, you cited to a paper by Keane and Berman, haven't you?
A. It may be true.
Q. Let's go to Exhibit 479, which was your report in the Daniels-Feasel case, Pages 9 and 10 of 94.
At the bottom of 9, it says, "For example, Keane and Berman in 2009 estimated that one-fourth of the observed

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increase in ASD prevalence in California between 1992 and 2005 was a direct consequence of diagnostic exchange between ID and autism."

Do you see that, sir?

A. That's what it says, yes.

Q. So you said that in the Daniels-Feasel case five years ago. Did you even cite to Keane and Berman in this report?

A. I don't recall whether I did or didn't.

Q. Well, is Keane and Berman even in your materials considered?

MS. BROWN: Objection to the form.

THE WITNESS: I don't know if it is or isn't.

MR. WATTS: Go to Page 30 of Exhibit 404, which is his materials considered list.

MS. BROWN: What is the exhibit?

MR. WATTS: 404. His

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materials considered list.

Page 30.

(Document marked for identification as Exhibit Kolevzon 404.)

BY MR. WATTS:

Q. If you cited to Keane and Berman, it should be right between Entry Number 2 and Entry Number 3, between Kim and Kristen -- or Kirsten, right? And it's not there.

A. It does not appear to be there.

Q. Can you explain for the ladies and gentlemen of the jury why, in 2018, you explained that only 25 percent of the increase is because of this; and in 2023, you just took out the reference altogether, suggesting something entirely otherwise?

MS. BROWN: I object. That completely misstates these documents.

THE WITNESS: So the

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opinions are the same. The trends are the same. The explanation is the same.

I've not included one reference that supports that.

BY MR. WATTS:

Q. Now, if Keane and Berman said changes in practices for diagnostic -- diagnosing autism accounted for only one-fourth of the observed increase in prevalence in California, that means three-quarters of the increase has to be explained by other factors, right?

MS. BROWN: Objection. Lacks foundation.

THE WITNESS: There are many other factors, many of which I've described here, which include change in diagnostic criteria, younger age of diagnosis. And other just cultural shifts in the acceptance of the diagnosis.

BY MR. WATTS:

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Q. Now, have you done any sort -- going back to 545, please.

Have you done any sort of work to be able to tell the ladies and gentlemen of the jury what the percentage of the rate increases explained by these five examples that you gave?

A. I don't think I'm able to do that, sitting here today.

Q. Well --

A. I think it's clear that all these factors have contributed to the rise in prevalence, for sure.

Q. You can't add it up to 100 percent, can you?

MS. BROWN: Objection.

BY MR. WATTS:

Q. Not even close.

MS. BROWN: Objection to the form.

THE WITNESS: So I haven't added it up. I haven't done a calculation.

The point here is that

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there's an enormous increase in the prevalence of autism. That the true increase -- the true -- the true incidence has not actually changed. And that much of the increase in prevalence can be accounted for by these factors.

BY MR. WATTS:

Q. Is it much of the increase, some of the increase, a small percentage of the increase, or all the increase?

A. I'm not going to guess.

Q. You don't know?

MS. BROWN: Objection to the form.

THE WITNESS: Was that a question?

BY MR. WATTS:

Q. Yep.

A. What's the question?

Q. You don't know what percentage of the increase of prevalence is accounted for by these supposed artifactual excuses you give versus

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environmental, do you?

MS. BROWN: Objection. Argumentive. Lacks foundation.

THE WITNESS: So I'm not sure that you can use the word "excuses" to characterize it, first of all.

I can't, at this moment, assign values, because this would probably require mathematical modeling that I'm not able to do, certainly not sitting here today.

BY MR. WATTS:

Q. Other people have done that mathematical modeling and concluded that there's got to be something else environmental going on, haven't they?

MS. BROWN: Objection to the form. Lacks foundation.

THE WITNESS: So it's clear that there are potentially environmental effects, because heritability is not 100 percent.

However, those environmental

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effects are, as of yet, basically unknown and certainly do not include acetaminophen.

BY MR. WATTS:

Q. Doctor, other people have done the work to figure out what percentage of the rate of prevalence increase is attributable artifact versus it's got to be environmental, haven't they? You've seen that?

A. So I would object to the "got to be environmental" part.

Q. Okay. Let me show you Exhibit 418 as one example.

(Document marked for identification as Exhibit Kolevzon 418.)

BY MR. WATTS:

Q. And this is an article written by Martha [sic] Cone in 2009, and I just want to give you a couple of quotes in this article.

At the bottom of the first page, top of the second --

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MS. BROWN: Well, he's not familiar with it. Let's let him read it first.

MR. WATTS: Split the page.

BY MR. WATTS:

Q. It quotes Martha -- Irva Hertz-Picciotto, an epidemiology professor at the University of California, Davis, who led the study. She says, "It's time to start looking for the environmental culprits responsible for the remarkable increase in the rate of autism in California."

This is a doctor that you have published with in the past, right?

MS. BROWN: Objection. Lacks foundation.

Can he read the article first.

BY MR. WATTS:

Q. Sir, have you published with her?

A. I may have.

Q. Okay. All we have to do is

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type in Picciotto --
A. Yeah.
Q. -- into the file of your publications, and we'll see it or not, right?
A. Of course.
Q. Okay.
And if we go to Page 3 of 6 it says, "The California researchers concluded that doctors are diagnosing autism at a younger age because of increased awareness. But that change is responsible for only about a 24 percent increase in children reported to be autistic by the age.
"'A shift towards younger age at diagnosis was clear but not huge,' the report says.
"Also, a shift in doctors diagnosing milder cases explains another 56 percent. And changes in state reporting of the disorder could account for around a 120 percent increase.
"Combined, Hertz-Picciotto

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says those factors 'don't get us close' to the 600 to 700 percent increase in diagnosed cases."
Did I read that correctly?
A. You're reading from a web-based article by somebody that I've never heard of, relying on information that I'm not able to verify.
Q. You've never heard of Dr. Hertz-Picciotto?
A. No. I have never heard of Marla Cone, who wrote this article.
Q. Okay. But the quote from Hertz-Picciotto, have you ever talked to her about her opinions in this regard?
A. I have not talked to her -- to her about it personally, no.
Q. And if we go farther down the page, she's quoted again. After she says they don't come close to getting us to the 6- to 700 percent increase, it says, "'There's genetics and there's the environment. And genetics don't change in such short periods of time,'

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Hertz-Picciotto, a researcher at UC Davis M.I.N.D. Institute, a leading autism research facility, said in an interview on Thursday."
Do you agree with that statement?
A. I agree with parts of that. And I think that it's clear there are both genetic, which makes up the vast majority of the cause of autism, and then, likely, some environmental risk factors that we have, as of yet, not reliably identified.
Q. Now, I want to talk to you about that last statement that you just made.
First of all, you have stated in many papers and depositions that autism spectrum disorder is a heterogenous condition, right?
A. Both clinically and genetically, yes.
Q. Okay. And for people who don't talk like doctors, heterogeneity

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means what?
A. There are a lot of different genetic causes and there are a lot of different symptoms.
Q. Okay. A lot of different environmental causes.
A. So, hypothetically. It's a speculation at this point, but it's certainly possible.
Q. Now, let's see if we can go about it this way.
Would you agree with me that if somebody fractures their femur, the fractured femur is a broken leg, right?
MS. BROWN: Objection. Incomplete hypothetical.
THE WITNESS: I'm not an orthopedic surgeon, but in layperson's terms I would assume that to be the case, yes.
BY MR. WATTS:
Q. The fractured femur is not the cause of the broken leg, it's the result, right?

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MS. BROWN: Objection to the form of the question.
THE WITNESS: Depends.
BY MR. WATTS:
Q. Well, if I put a bat to your femur -- and I wouldn't do that to you -- but if I did and I broke your femur, the swinging of the bat is the cause of your broken leg, right?
MS. BROWN: Objection to the form of the question.
THE WITNESS: It depends.
BY MR. WATTS:
Q. Well, autism, does it result from a gene change that occurs?
MS. BROWN: Objection. Vague.
THE WITNESS: The only commonly accepted cause of autism today, as we understand it, is genetic in origin.
MR. WATTS: Objection. Nonresponsive.
MS. BROWN: Object.

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BY MR. WATTS:
Q. Do you know what the phrase "ipsi dixit" means?
MS. BROWN: Objection to the form of the question.
THE WITNESS: I do, but only because I read a deposition yesterday.
BY MR. WATTS:
Q. Okay.
MS. BROWN: You guys are asking the same questions.
BY MR. WATTS:
Q. Did you read --
A. And I took Latin in high school.
Q. Yeah. Did you read "Alice in Wonderland"? It is, because I say it is.
A. Yes.
MS. BROWN: Objection to form.
BY MR. WATTS:
Q. And so let's -- let's probe

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on this, "It is, because I say it is."
Does autism ever happen without a change in genetic composition or gene expression?
MS. BROWN: Objection. Calls for speculation.
THE WITNESS: So to the state of our knowledge today, the only reliable, replicated, consistent causal factor in autism is genetic in origin.
BY MR. WATTS:
Q. Okay. That wasn't my question.
The change in the gene is the broken leg, right? It's the consequence that manifests itself through clinical conditions that you call autism, right?
MS. BROWN: Objection to the form of the question. Lacks foundation. Vague.
THE WITNESS: I think you need to clarify the question.

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BY MR. WATTS:
Q. Well, let's see if we can go about it this way.
There are all sorts of study about what causes that change in the gene, right?
MS. BROWN: Objection. Vague.
THE WITNESS: There are some studies that explore what are called epigenetic factors. Other studies that very, very clearly document that de novo, spontaneous, what are called stochastic mutations occur at the time of conception.
BY MR. WATTS:
Q. Okay. So we're going to get into all of that, but let's keep it on a global fifth-grade level.
Is there any, according to you, any case of autism that exists without a modification of the gene of some sort?

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MS. BROWN: Objection. Vague. Calls for speculation.
THE WITNESS: The current understanding is that the only accepted cause of autism is genetic in origin.
BY MR. WATTS:
Q. And we're almost getting there.
When you say genetic in origin, something happened to the gene --
A. Yep.
Q. -- that altered it, right?
MS. BROWN: Objection. Vague.
THE WITNESS: So by "something," what do you mean?
BY MR. WATTS:
Q. Well, we're going to get there.
But in order to have autism, you have to have a changed gene from what's normal, right?
MS. BROWN: Objection.

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Lacks foundation.
THE WITNESS: I mean, it could be sort of a common variation that might manifest as normal in one individual, but as it's inherited and interacting with other genes, could be pathologic in another individual.
BY MR. WATTS:
Q. See if can go about it this way.
I do a lot of pesticide work.
A. Yeah.
Q. Tell the jury what a clastogen is.
A. I'm not a pesticide expert. I have no idea.
Q. You are not aware of what a clastogenic substance?
A. A what?
Q. A clastogenic substance.
A. Clastogenic? No --
Q. Clastogenic,

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C-L-A-S-T-O-G-E-N-I-C.
A. I think that's well outside of my area of expertise.
Q. Okay. If I tell you that it's a mutagenic agent that disturbs normal DNA-related process, causes DNA strand breakages, deletion of entire chromosome sections, insertion of chromosome sections, rearrangements of chromosome sections, does that make a lot of sense to you?
A. All of that make sense.
MS. BROWN: Objection. It lacks foundation.
BY MR. WATTS:
Q. Okay. So a clastogen is something that alters the genetic presentation, right?
MS. BROWN: Object. Lacks foundation.
THE WITNESS: So, again, I don't know what a clastogen is, so it's hard for me to comment about it.

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But what you asked me was, did that string of words make sense.
BY MR. WATTS:
Q. Yeah. And it did, didn't it?
MS. BROWN: Let him finish.
THE WITNESS: Those -- those string of words make sense. There is the possibility.
BY MR. WATTS:
Q. Okay. There are environmental products that are clastogenic because they can alter the general metallic presentation in the way that I just described, right?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: This is, again, outside of my area of expertise.
BY MR. WATTS:
Q. Okay. But in that situation, if you have a substance, when

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somebody is exposed to it, it results in their change of their genetic presentation. The substance caused the change in the genetic presentation; agreed?

MS. BROWN: Object. It lacks foundation. It's an incomplete hypothetical, and the doctor has already said it's outside his area of expertise.

THE WITNESS: So I'm happy to talk about this as it relates to autism, which I think is what my area of expertise is and what I came here to testify about.

BY MR. WATTS:

Q. Okay. I really need you to answer my question.

MS. BROWN: He did.

BY MR. WATTS:

Q. If a pollutant, for example, is a clastogen and results in an interruption of the genetic presentation, either deletion, insertion, the

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rearrangement of entire chromosome sections or DNA strand break. That substance is the cause of the change in the genetic presentation; would you agree with me?

MS. BROWN: Object. It lacks foundation.

He already said he doesn't know what a clastogen is.

MR. WATTS: Well, that's shocking.

THE WITNESS: I don't feel comfortable testifying about the structural damage that pollutants may or may not cause.

BY MR. WATTS:

Q. Okay. And part of the reason, you don't even know what a clastogen is, do you?

A. I have never heard that particular term before.

Q. I am frankly shocked.

MS. BROWN: That's inappropriate, and you know that,

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Mr. Watts.

Let's behave.

BY MR. WATTS:

Q. Let's -- let's get boned up on that before trial, okay?

MS. BROWN: That's not appropriate.

BY MR. WATTS:

Q. Now, let me ask you this. When you don't know what caused the change in gene presentation, you all call that idiopathic autism, right?

MS. BROWN: Objection to the form.

THE WITNESS: Idiopathic autism is defined as autism where we don't have an identified gene, yeah.

BY MR. WATTS:

Q. And over time, in your testimonies in the last five years, you've said in the Huddleston deposition, "80 percent of the time there is no specific gene identified as the cause,"

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right?

A. I think at the time of that deposition, that was probably the correct number. That number changes over time.

Q. Okay. And to be fair, more recently you said it's more like 70 percent of the time we don't know what caused it, right?

A. That's probably a more accurate reflection of where we are today.

Q. Okay. And to your credit, and others studying it, we're learning as we go, right?

A. We are in an exponential phase of discovery, yes.

Q. All right. Now, let's talk about the concept of heritability versus de novo genetic changes, okay.

First of all, from the standpoint of common variants, are common variants, by definition, inherited variants?

A. Common variants are thought

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to be inherited variants, yes.
Q. Okay. And with respect to the common variants, we can use something called monozygotic twin studies to look for concordance between the two twins in terms of how often they have autism, both of them, versus one of them, right?
A. That's how you define heritability, yes.
Q. Okay. Now, let me give you an example of that. And we're just going to go through this step-by-step.
Exhibit 438 is a paper by Gaugler that you cite in your report.
A. Yeah.
(Document marked for identification as Exhibit Kolevzon 438.)
BY MR. WATTS:
Q. And if we could go to Page 3 of Exhibit 438.
And Gaugler --
A. Sorry, this is a press release from Mount Sinai about this

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paper. And all I have is a bunch of papers. I don't have the actual...
MS. BROWN: Is this 438?
MR. WATTS: Yeah, I think you're --
MS. BROWN: I gave you 435. That's my fault. Here you go.
MR. WATTS: Okay. Strike what he just said. He didn't mean it.
BY MR. WATTS:
Q. I get it, you were given the wrong document.
Now we're on 438, let's talk together. This is the Gaugler manuscript entitled, "Most Genetic Risks For Autism Reliance on Common Variation," and it's in the NIH Public Access author manuscript, right?
A. Yes, from 2014.
Q. From 2014.
And I want to take you to Page 3, and part of what he says on Page 3 is "A recent, large study of twins

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places heritability at 38 percent."
Do you see that?
A. Yep.
Q. Now, he cites to Footnote 20; is that right?
A. Yeah. It's probably the Hallmayer study.
Q. I'm sorry? Say what you said.
A. It's probably the Hallmayer study.
Q. Okay. And when was the Hallmayer study?
A. Yeah. 2011.
Q. Okay. And that's a study with which you are familiar?
A. I am.
Q. And the Hallmayer study placed the heritability of autism at 38 percent, right?
A. The Hallmayer study was one among 14 or 15 studies that had overblown estimates -- or underblown underestimates of heritability.

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Q. That wasn't my question. My question was, the Hallmayer study placed heritability at 38 percent, correct?
A. I think that they looked at different ways of measuring, and, yes, one of the estimates was 38 percent.
Q. And it's being cited by Gaugler, which is one of the papers that you cited in your report, right?
A. Yes.
Q. Okay. Now, you, saying that Hallmayer was low, have more recently said that common variations contribute to about half of the autism risk, right?
A. That's what it's thought of, yes.
Q. Okay. So by virtue of that statement which you've said several times, to be fair to you, your position is, is that half of the risk of autism is inherited from parents; is that right?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: So this is

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complicated, because --
BY MR. WATTS:
Q. Let me re-ask the question.
MS. BROWN: Can we --
BY MR. WATTS:
Q. I see why you're saying it's complicated. Let me see if I can go about it this way.
You remember how we talked about 20 or 30 percent are genes that we know and the rest we don't know?
Did you mean to say that of the genetic source that we have, half of that is common variation?
A. About that, yes.
Q. Okay. And common variation is, in effect, inherited variation, right?
A. Yep.
Q. Okay. Now I want to play you a video that I think does it pretty well from the standpoint of Dr. Chung, and I think there was an intention to play this for her on Wednesday. But I

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want to ask your thoughts about it.
Okay. Give me just a second.
(Document marked for identification as Exhibit Kolevzon 549.)
MS. BROWN: We're playing a video for Dr. Kolevzon of Dr. Chung.
MR. WATTS: We absolutely are doing that.
MS. BROWN: All right. I'm going to object as lacking foundation, as I suspect Dr. Kolevzon hasn't seen it before.
MR. WATTS: Okay. Objection overruled. We're going to keep going -- I'm kidding.
BY MR. WATTS:
Q. Exhibit 549. Just play the video, and I'll ask your thoughts about it.
TRIAL TECH: I'm sorry,

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which one?
MR. WATTS: Exhibit 549.
(Video played.)
DR. CHUNG: You take two identical twins, and you ask yourself, if one twin has autism, what is the probability that the other twin has autism? If this were genetic, if it were entirely genetic, what would you think that answer should be?
100 percent. Absolutely.
Is that number up there 100 percent? Absolutely not. 77 percent. Some people have estimated 80 percent. But it's not 100 percent.
So there's some difference that not, at least not in the genes that happened right at the time of fertilization. But there's some change, some difference between the two of them. That number is high,

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though.
So as an example, as a comparison, now, if you take fraternal twins, right. So these are cases that are like brother and sister, but they share the same in utero environment. They can be in the same household. They share the same -- some of the same postnatal factors. And if you look at that, they share 50 percent of their genomes, or 50 percent of their genetic information. The concordance rate between those is 31 percent.
But now take as an interesting comparator to that, siblings. So these are, again, genetically sharing 50 percent of their genetic information, but they don't share the same in utero environment. They don't necessarily share the same exposures after they are born.

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And in that particular case, it's lower. Right. Statistically, significantly lower, only 20 percent in those cases.

And in the general population, as you've heard, it's about 1 percent, or, these days, 1 in 59 individuals with autism.

So what does that tell me? What that tells me is, there are some genes. I don't -- doesn't tell me what the genes are, but it tells me genes are important in that, but it's not all in the genes, right.

Part of it is genes, part of it is something else.

(Video playback ended.)

BY MR. WATTS:

Q. Her description -- and I played it because it's all very compact -- about the difference in autism rates between identical twins, lower in fraternal twins, lower than that in

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brothers and sisters, you agree with that as a concept, right?

MS. BROWN: Objection.

THE WITNESS: Yes.

BY MR. WATTS:

Q. Okay. Now, you said Hallmayer was low when it said that only 36 percent is nonheritable, right?

A. 30 -- I said it was low and 36 percent was heritable.

Q. Yes, I'm sorry. Yes, I misstated.

You have in a paper since then said that 36 percent is inherited, right?

MS. BROWN: Objection to form.

THE WITNESS: Say it again.

BY MR. WATTS:

Q. Well, let me just show it to you. Exhibit 434.

(Document marked for identification as Exhibit Kolevzon 434.)

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BY MR. WATTS:

Q. Entitled, "Convergence of Genes and Cellular Pathways Dysregulated in Autism Spectrum Disorder," published in The American Journal of Human Genetics, Volume 94, Issue 5, of May 1st, 2014. The first author is Dalila Pinto.

Do you see that?

A. Yes.

Q. And if you go about halfway down all the -- all the authors?

A. Yeah, I know. This is -- I know what this is.

Q. Okay. There is your name, correct?

A. Yep.

Q. And if we go to Page 15 of 43, and we highlight, it says, "Inheritance data showed that 64 percent of pathogenic CNVs were de novo events" -- and we'll get to that in a second -- "and the remaining (36 percent) were inherited."

Do you see that?

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A. Yes.

Q. Okay. Now, because you put this in the same sentence --

A. But --

Q. Let me just say, what is a de novo event, first of all?

A. All right. So to clarify, when it says inherited here, that's many -- that's among the things that are considered to be heritable.

De novo just means that it occurred spontaneously, so it was not specifically passed on from a parent. So the common variations exist in parents but are not causing any clinical symptoms.

Q. Now --

A. When they are inherited to the child, they can interact with other genes and cause clinical symptoms, which is different than other heritable genetic factors which can occur within the sperm or the egg. So they are still essentially passed on, right. They are

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just not common variations.
So they can either be de novo, rare variations, or even some de novo, rather, rare inherited variations.
So this 36 percent represents the universe -- a piece of the universe that is considered to be heritable.
Q. Okay. Now, here is my question. I think it was about what is a de novo gene mutation --
A. Just means spontaneously occurred.
Q. Okay. Go back to my example about the clastogenic chemical that alters a gene presentation.
Does that go in the de novo category?
A. Generally speak --
MS. BROWN: Objection to the hypothetical.
Go ahead.
THE WITNESS: When it comes to autism, the de novo mutations

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that we've identified, that we know cause autism, occur at the time of conception.
BY MR. WATTS:
Q. I'm asking, if that mutation which results from environmental exposure -- I'll give you that it happens at the time of conception -- is that in the de novo category?
A. Can you repeat the question?
Q. When you talk about de novo mutations, those are nonheritable, right?
A. No. De novo mutations are heritable.
Q. Well, what about ones from the environment, are they de novo?
MS. BROWN: I object to the question. It doesn't make sense.
THE WITNESS: I don't know what you mean by ones from the environment.
BY MR. WATTS:
Q. Well, when you use the phrase "de novo," does it include

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environmentally caused increase in risk because of a gene mutation that an environmental agent caused, yes or no?
MS. BROWN: Objection. Vague. Lacks foundation.
THE WITNESS: So heritability is defined by the concordance or discordance between monozygotic and dizygotic twins.
BY MR. WATTS:
Q. Done that. Now de novo.
A. De novo mutations in the case of autism that have been identified up until now occur when sperm meets egg.
Q. I understand when it occurs. I'm saying why it's occurring.
A. So the current state of our understanding, that these things are what are called stochastic; they are random.
Q. So we're back to idiopathic autism. You just don't know what's causing it yet, is what you're saying.
A. No, we know what's causing it. There's the gene that's causing it.

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Q. Well --
A. Why does the gene mutate? We don't necessarily know.
Q. But there's strong evidence that nonheritable, pre- and perinatal events are likely to also have an etiological role?
MS. BROWN: In autism?
MR. WATTS: Yep.
THE WITNESS: So in autism, because the heritability is not 100 percent, there are likely environmental events that play a role, they just are, as of yet, unidentified.
And bringing it back to this case, when you look at the literature around acetaminophen, it's certainly not considered a risk factor.
BY MR. WATTS:
Q. Well, look, let's go back to 2011, before you were ever hired in this case, and let's look at the first edition

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of the Textbook of Autism Spectrum Disorders.

MR. WATTS: Exhibit 422.

BY MR. WATTS:

Q. And if we go to Page 239. You, Reichenberg, Gross and Susser write, "Parental and Perinatal Risk Factors for Autism."

And the very second sentence in your book chapter is, "Yet there is strong evidence that nonheritable pre- or perinatal events are also likely to have an etiological role," right?

A. Because heritability is not 100 percent, there are going to be nonheritable events that likely play an etiological role.

Q. And in the book chapter that didn't make its way into your CV, you said the same thing 11 years later in the second edition, right?

MS. BROWN: Objection to the form of the question.

THE WITNESS: I'm sure I

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said something similar, because it continues to be true.

BY MR. WATTS:

Q. And an etiological role means what?

A. Causal role.

Q. Okay. So a prenatal event means what?

A. Prenatal just means before the time of birth.

Q. And perinatal means during the time of birth, right?

A. Birth and delivery, yeah.

Q. And so, yet, there is strong evidence that nonheritable before-the-time-of-birth or during-the-time-of-birth events are likely to also have a causal role.

That's what that sentence means, right?

A. So, theoretically, because heritability is not 100 percent, there are likely going to be prenatal and perinatal events that play a causal role.

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Q. And that sentence doesn't use the word "theoretically" at all, does it? It says there is strong evidence that it's true.

A. Well, it's -- there's strong evidence it's true, based on twin studies that show that heritability is not 100 percent.

Q. Now, Doctor, I want to play you another video from Dr. Chung, Exhibit 549, and get your comment on it.

MS. BROWN: And same objections to the Dr. Chung videos.

And could we also, Counsel, identify them for the record? Dates, where they came from?

MR. WATTS: Sure. This one is December 16, 2018.

MS. BROWN: Okay. And for the other one, we can go back and fill it in, please.

MR. WATTS: Yeah, I'll do that.

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MS. BROWN: Thank you.

(Video played.)

DR. CHUNG: Two identical twins, and you ask yourself if one twin has autism, what's the probability that the other twin has autism? If this were genetic, if it were entirely genetic, what would you think that answer should be --

MS. BROWN: We just watched this one.

THE WITNESS: Same clip.

DR. CHUNG: 100 percent. Absolutely. Is that number 100 percent? Absolutely not. 77 percent. Some people have estimated 80 percent. But --

(Video playback stopped.)

MR. WATTS: Stop. Had a glitch. Sorry.

548, please.

(Document marked for identification as Exhibit

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Kolevzon 548.)
(Video played.)
DR. CHUNG: The bottom line is, for any one person that I see in terms of identifying a cause, I can come up with the answer these days about 20 percent of the time. That's the good news/bad news. So you can now -- that's the take-home message. You can go home now if you're bored.
So with that, though, that means 80 percent of the time we have not yet figured this out. It's not all about the genes, but for the majority of individuals where we can pin it down to one thing, it is in the genes.
But we know there are other factors from epidemiological studies, which I won't have time to talk about. We do know that there are infections during pregnancy. We know there are

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infections in early childhood that can cause this. Whether it's the virus itself, or whatever might be infected; whether it's some of the inflammatory response or what we do to fight the infection. It could be some of both.
There may be things in our environment, so things that we're exposed to. Things like pollutants, chemicals, things that are changing in that.
But we don't know all the answers, is the bottom line.
(Video playback ended.)
BY MR. WATTS:
Q. Do you agree with what she says?
A. You need to be more specific.
Q. Okay. Things like environmental pollutants. Do you agree with her?
A. I don't think it's been

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established that environmental pollutants are a cause of autism, no.
Q. Let's -- let's go to the identification of autism genes.
Inherited or common variants account for 50 percent of the genetic risk. We've established that, right?
A. Yes.
Q. Give us examples of the common variants that have been identified.
A. So off the top of my head, I don't have a lot of good examples of common variations. There's probably, you know, five or six of them. The problem is that they are common, and they have a very weak effect. And that it's only through interacting with many, many of them are you going to produce sort of a perfect storm of autism.
Q. You said something I agree with, that they have a weak effect. Why is it that common variants have a weak effect, whereas de novo variants are more

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powerful?
A. Because common variants that are inherited from parents are not having any kind of penetration. They are not creating an actual symptom. They are sort of impairing, perhaps, the way that a protein functions. But in and of itself, it is not enough to produce, like, a clinical picture.
When it's inherited and then combined with many, many other common variants, that's when it occurs.
Q. Okay. So a common variant by itself is weak and hard to clinically diagnose, agreed?
A. In general that's the case.
Q. Common variants become clinically diagnosable when they are inherited, when they are working together with other common variants?
A. Yes. But even then we have not been super successful at identifying common variants.
Q. And so common variants that

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are interacting with de novo variants, does that present, clinically, in a way that you have a more powerful or more easily diagnosable case of ASD?

MS. BROWN: Objection to the form.

THE WITNESS: That's sort of a hypothetical. I think when you have rare de novo variants that are highly pathogenic, and in a single gene are sufficient to cause autism, then you have common variations that require interaction with multiple other genes in order to produce a clinical picture.

BY MR. WATTS:

Q. Now, this is my last Chung video, I promise. Exhibit 550.

(Document marked for identification as Exhibit Kolevzon 550.)

MR. WATTS: This one is from April 2nd of 2021.

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(Video played.)

DR. CHUNG: So there are two distinctly different -- when I think of genetics of autism, there are sort of two different buckets that I think of. So within -- and let me try and point this out.

So we return about -- to about 10 percent of SPARK families currently, about 10 percent of you have a genetic finding that we see that we're very certain is the -- a major contributor to the autism in your family. And so we return that information to you.

For most of the individuals, not all, but most of the individuals that we currently recognize that in, those are genetic changes that are de novo, or new, in the individual with autism.

And so what I mean by that is it's not something that's in

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the mother's sample that was sent in, it's not something in the father's sample that was sent in that we see, then it seems to have started first with the individual with autism.

So that's genetic, but it was not inherited.

So it didn't come down from mom or dad. But it is genetic in the individual with autism.

(Video playback ended.)

BY MR. WATTS:

Q. Do you agree with that?

A. Yes.

Q. Okay.

A. It's still different than the heritability, though.

Q. Well, she says it's genetic but not inherited. That's what she said, right?

A. It wasn't passed from the parent to the child, but it occurred in the parents's gamete, the sperm or the

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egg.

Q. Now, on Page 23, Paragraph 51, of your report, Exhibit 403, you say, "Rare genetic variants are often de novo (i.e., not inherited from the mother or the father), highly penetrant (i.e., expressing associated trait), and considered to be causal to autism spectrum disorder," right?

A. That's correct.

Q. Okay. And then one last thing with respect to Chung, PowerPoint Exhibit 568, on April 25, 2023, Slide 29.

She says, "De novo variants have a strong effect on the presence of autism." Whereas "common/mild effect variants have milder effects."

Do you agree with that?

A. It depends, but, in general, that's a true statement.

Q. Do you know why it's true?

A. I think we've gone over this.

Q. And why is it true again?

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A. So common variations by themselves don't produce clinical effects, and only when they are interacting with many other common variations do you actually see, like, a threshold met, essentially.
Q. Okay.
A. And they're -- they are common.
Q. Okay. And they are common, and there's five or six of them, and you just can't name any of them today?
A. No. There's hundreds, if not thousands of them.
Q. I'm talking about common variants.
A. There's hundreds --
Q. You said -- you said three minutes ago there were five or six of them.
A. No. What I said is there are five or six that have been reliably identified as of yet, based on the current state of science. But there are

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likely hundreds, if not thousands, of common variants that contribute to autism.
Q. Okay. As to the ones that we've identified, five or six, can you list any of them?
A. Not off the top of my head, no.
Q. Not one?
A. No, I don't...
Q. Okay. Now, let's talk about the last 15 years of science.
Back in 2007, in Exhibit 414, you said, "To date there are no known genes for autism."
A. Sorry. Can you -- where are we?
Q. Yeah. Exhibit 414, Page 331. And I'm talking about the time between then and now. And I understand why, I just wanted to start with a baseline.
Do you see that statement on your paper in 2007, "There are no known

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genes for autism"?
A. No.
Q. Look at the screen.
MS. BROWN: Where is it in the article?
THE WITNESS: What page are we on?
MR. WATTS: We're on 44 --
MS. BROWN: Conclusions.
MR. WATTS: We're on Page 331.
THE WITNESS: 331.
BY MR. WATTS:
Q. "To date, there are no known genes for autism."
Do you see that?
A. Yeah. Even in 2014, I would disagree with that statement.
Q. Okay. But this is in 2007.
A. Fragile X.
Q. Okay. Now, let's go forward in time. In 2011, in your first edition of the Hollander book, Exhibit 422, Page 244.

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MS. BROWN: This one.
THE WITNESS: 442? Sorry. 442?
BY MR. WATTS:
Q. Yep.
No. Exhibit 422, Page 244, and there it is on the screen.
You say, again, in 2011, "Because currently there are no known" -- "there are no established risk genes for autism."
Do you see that?
A. Yeah. I see that on the page, yeah.
Q. Okay. And then in 2013, if we can go to Exhibit 430.
(Document marked for identification as Exhibit Kolevzon 430.)
BY MR. WATTS:
Q. In the book, Neurobiology of Mental Illness, Fourth Edition, edited by Charney, published in 2013, you were the first author in a book chapter, 77?

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A. Yep.
Q. Entitled "Autism Spectrum Disorders"?
A. Yes.
Q. And if we go to Page 1027 of that. The upper right-hand corner.
The bottom of the last sentence in the upper right-hand corner, it says, "One important conclusion from the empirical and theoretical data is that many, and even most, of the candidate gene association studies published in autism spectrum disorder are very likely false-positive findings."
A. Mm-hmm.
Q. If you could, explain why that's true.
A. So I think that when you look at, like, these snips that are common and have weak effects, and you use things like linkage dysequilibrium studies and see that they are more prominent in people with autism, they don't hold up in larger datasets. So you

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just get spurious effects.
Q. Okay.
A. Which is totally in contrast with the rare variants.
Q. Now, since 2007, the technology available to you to be able to look into the DNA and the gene expression has exponentially improved, agreed?
A. The technology and the analytic methods, yes.
Q. Okay. So let's talk about the technology first.
Since about 2007 you all have used chromosomal microarrays, correct?
A. Yes.
Q. And that allowed you to see the deletion or the duplication or the reordering of genetic code, right?
A. So, yeah, to a certain extent of resolution, depending on how many probes, yes, that's basically true.
Q. Okay. And in about 2013-'14, you've got something even

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better, called exome sequencing, right?
A. Right.
Q. And beginning around 2013, that would be kind of the next generation of higher resolution testing, right?
A. Yes.
Q. And that got the technology even better, where you can see more than you could with just microsomal arrays, right?
A. Generally that's true.
Q. Okay. We've got other techniques since then. I mean, in the earlier says you had karyotyping, right?
A. True.
Q. Then you had high-resolution karyotyping, right? Yes?
A. Yes.
Q. And then you had something called FISH, fluorescent in situ hybridization.
A. Yes.
Q. And then you had the chromosomal microwave beginning about

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2007?
A. Yes.
Q. And then now we have something called WES, what is that?
A. Whole exome sequencing.
Q. Okay. And even today, with all that technology, each of these rare variants account for only about 1 percent of autism or less, right?
A. So given rare variant, probably only accounts for 1 to 2 percent in a given case.
Q. Okay.
A. When you add up all the rare variants, it accounts for much more.
Q. Okay. Let's talk about Exhibit 522. This is a video that you gave. It's on YouTube, and I will tell you it's undated. I couldn't find the date.
But let me just play it and ask whether you still agree with it. Maybe you can tell me when it was.
(Document marked for

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identification as Exhibit Kolevzon 522.)

(Video played.)

"DR. KOLEVZON: To date all the genetic defects reliably identified to cause autism are rare variants, each accounting for only about 1 percent of autism or less.

However, when these rare genetic defects are present, they are always associated with clinical symptoms, which more often than not include autism."

(Video playback ended.)

BY MR. WATTS:

Q. Okay. So is that still true today, that even the most common rare variant is about 1 percent of the total risk for autism?

A. So 1 to 2 percent. That's probably true.

Q. Okay. Let's talk about some of the more spoken of rare variants.

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Fragile X syndrome is a rare variant, right?

A. Right.

Q. It's caused by mutations in the FMR1 gene that results in the loss of the FMR protein, which is critical for brain development, right?

A. Yes.

Q. You have testified that it's the most commonly known rare variant cause of autism spectrum disorder, right?

A. I think that that was probably old testimony.

Q. Okay.

A. But it's certainly among the most commonly known.

Q. Now let's take fragile X syndrome and call it our broken leg.

Do you know what the epidemiological research has demonstrated causes fragile X syndrome?

MS. BROWN: I'm just going to object to the hypothetical about the leg.

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MR. WATTS: That's a great example.

BY MR. WATTS:

Q. Keep going.

A. Fragile X syndrome is essentially inherited from mothers.

Q. Have you looked at any epidemiological work on fragile X syndrome?

A. I did not investigate that specific question today.

Q. Let me put up Exhibit 547, just to give you an example. One of many.

(Document marked for identification as Exhibit Kolevzon 547.)

BY MR. WATTS:

Q. There's a lot of work's been done on what causes fragile X syndrome?

A. Well, it's CGG repeats, inherited from a parent.

Q. And let -- let's use --

MR. WATTS: Objection.

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Nonresponsive.

MS. BROWN: Object.

I don't have 547, I don't think. Oh, wait.

MR. WATTS: I'm pretty sure you do. It's got my handwriting on it, on the back.

MS. BROWN: Well, we go from 546 to 548. Let me try the other box.

MR. WATTS: Here, take mine. Give it back, though.

MS. BROWN: Oh, hold on. 546 is 547. Got it.

MR. WATTS: We're good.

MS. BROWN: We got it.

BY MR. WATTS:

Q. Okay. This is a study by Saldarriaga. "Increased severity of fragile X spectrum disorders in the agricultural community of Ricaurte, Columbia."

A. Hold on. Let me just read it for a second.

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Q. Do you see in the abstract they said, "We found an increased frequency and severity of symptoms of fragile X spectrum disorders, which might be related to the vulnerability of the FMR1 premutation carriers to higher exposure to neurotoxic pesticides in this rural community"?

MS. BROWN: Object as lacking foundation.

THE WITNESS: So these are -- these are not people with fragile X syndrome. These are premutation carriers. These are the parents that when you pass on, it becomes a full mutation.

BY MR. WATTS:

Q. Did you cite -- I'm sorry.

A. And then you have fragile X syndrome.

Q. Did you cite to any studies that analyze whether the prevalence of fragile X syndrome was higher in areas with pesticide exposure than not?

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MS. BROWN: Objection. Assumes facts. Lacks foundation.

THE WITNESS: My task here was to evaluate the relationship between acetaminophen used during pregnancy and autism, not fragile X syndrome.

BY MR. WATTS:

Q. Yeah, but you said it was genetic, not environmental. Have you looked at anything to say whether it's environmental or not?

MS. BROWN: Object to the form of the question.

THE WITNESS: Can you repeat the question?

BY MR. WATTS:

Q. Have you looked at any studies that have analyzed whether the prevalence of fragile X syndrome is based on exposure to things like pesticides or other environmental toxicants?

MS. BROWN: Objection to the form.

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THE WITNESS: So I have not.

BY MR. WATTS:

Q. Okay.

A. But this -- this particular study doesn't necessarily suggest that.

Q. Let's go to a different one, Phelan-McDermid syndrome. You've studied that in SHANK3 mice a lot, right?

A. I've spent a lot of time studying Phelan-McDermid syndrome, yes.

Q. You began studying in about 2009?

A. Sounds about right.

Q. You've looked at different pathway activity, hoping to either inhibit or decrease the activation so you can have an effect on the phenotype, right?

A. I collaborate with people that do that on the preclinical trial, and I do clinical trials in kids, yes.

Q. And you use SHANK3. You are using mice, right?

A. I don't work with mice

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myself. I collaborate with basic scientists who do.

Q. Yeah. Let me show a video, Exhibit 472, just as an example.

(Document marked for identification as Exhibit Kolevzon 472.)

(Video played.)

DR. KOLEVZON: You can actually stimulate mice with the missing copies of the SHANK3 gene in this case where you basically ring, like, a very loud bell and you can induce a seizure. These are called audiogenic seizures. And, remarkably, all of these phenotypes were rescued under the influence of this drug AMO-1.

Okay. And so for us, that was, like, you know, kind of an important sort of proof of concept, that there could be some real relevance to some kids with

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Phelan-McDermid syndrome.
(Video playback ended.)
BY MR. WATTS:
Q. That's your voice, right?
A. Yeah.
Q. Okay. Phelan-McDermid syndrome also goes by SHANK3 deletion syndrome, right? It's the same thing?
A. It's -- they're -- yeah. I mean, it's complicated. But sort of.
Q. If you want to get really in the weeds, it's the 22q13.3 deletion syndrome?
A. You can have any deficiency, or what's called haploinsufficiency of SHANK3, gives you the diagnosis of Phelan-McDermid syndrome. That can be through deletions, or it can be through single base para-sequence variants.
Q. Sure. But it's all about a loss of a small piece of what, Chromosome 22?
A. Small or large, yeah.
Q. Okay. And Phelan-McDermid

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or SHANK3 is about one-half to 1 percent of the rare variant autism cases, right?
A. Probably up to 2 percent when you include intellectual disability, but yes.
Q. And have you read any studies with respect to the relationship between environmental exposures and the prevalence of Phelan-McDermid syndrome?
MS. BROWN: Objection. Assumes facts. Lacks foundation.
THE WITNESS: So I'd be interested in studies that you have to show me if I haven't read them.
But the Phelan-McDermid syndrome is a de novo spontaneous mutation that occurs at the time of conception.
BY MR. WATTS:
Q. What was the answer to my question?
MS. BROWN: He answered it.
THE WITNESS: I said I'd be

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interested in any studies that you have to show me that the prevalence of Phelan-McDermid syndrome increases with environmental exposures.
BY MR. WATTS:
Q. I know you desire that. I'm asking, before you showed up here, have you bothered to look?
MS. BROWN: Well, that's argumentive. He answered your question, and he'll answer it again.
Go ahead, Doctor.
THE WITNESS: I've looked at every paper that has ever come out with Phelan-McDermid syndrome as it relates to clinical features.
It's possible --
BY MR. WATTS:
Q. What about etiology?
MS. BROWN: Let him finish.
THE WITNESS: Also, the etiology of Phelan-McDermid

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syndrome is SHANK3 haploinsufficiency.
BY MR. WATTS:
Q. Broken leg?
A. Excuse me? Broken leg?
Q. We've got a fractured femur that caused the broken leg again.
MS. BROWN: Again with the leg. I'm going to continue to object to the leg.
THE WITNESS: So -- yeah, the leg is an interesting analogy. I mean, let's think about the leg for a second.
If you have Phelan-McDermid syndrome -- and I'll pull your analogies together just for fun.
BY MR. WATTS:
Q. Cool.
A. And you are more prone to hyperflexibility, and you find yourselves in situations where you may have a fracture because you have fallen, because you've got motor skill deficits. And so

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in that case, it's not the bat, it's the Phelan-McDermid syndrome that caused the fracture.

Q. Let's look at 546, a paper by Boccuto.

A. Boccuto?

(Document marked for identification as Exhibit Kolevzon 546.)

BY MR. WATTS:

Q. Yeah. Are you ready?

A. Yes.

Q. "Phenotypic Variability in Phelan-McDermid Syndrome and Its Putative Link to Environmental Factors."

Have you seen this before?

A. Yes.

Q. Was it listed in your materials considered in this case?

A. I don't know if it was or wasn't.

Q. Okay. If we could go to Table 1, it's got "Possible contributions of environmental factors to

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Phelan-Mcdermid syndrome clinical presentation."

And it lists different environmental factors, including drugs and inflammations -- inflammation, and the like, right?

A. Yes.

Q. Okay. And it's got a bunch of references that talk about those contributions of environmental factors to Phelan-McDermid syndrome, right?

A. Yes.

Q. Okay. Doctor, with respect to your work with SHANK3 mice, are you doing work with respect to etiology or cause of the syndrome as opposed to treat it, in fairness to you?

A. So by point of clarification, I don't work with the mice. I collaborate with people who work with mice.

Q. Fair enough.

A. The etiology of Phelan-McDermid syndrome has been

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established. It's due to SHANK3 haploinsufficiency.

Q. Broken leg.

MS. BROWN: Again, with the broken leg. I'm going to object.

BY MR. WATTS:

Q. Go ahead.

A. And my role is always to try to develop ways to support or treat children.

Q. Okay. Let me give you some kudos and play Exhibit 469 for a second. It's one of your videos, and I just want to put it in the record.

MS. BROWN: Can you give us the date.

MR. WATTS: February 28, 2018.

(Document marked for identification as Exhibit Kolevzon 469.)

(Video played.)

DR. KOLEVZON: We create different kinds of model systems,

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whether it's mice models or rat models or even now, neurons derived from humans, and using iPSCs, or pluripotent stem cells, as it's called.

We use those models to really better understand the pathophysiology, and in this case, what's going wrong in terms of the nerve cell connections when SHANK3 is -- when there's loss of function of SHANK3.

Then we can test specific medicines in the models. And then if those medicines work at reversing either the behavior of the models or the way the nerve cells connect, for example, then we bring those medicines to clinical trials in kids.

And that's the pathway that we've been following.

(Video playback ended.)

BY MR. WATTS:

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Q. Is that one of the videos you did?
A. Yes.
Q. Okay. So you're working with people who are using the SHANK3 mice to help try to come up with a clinical treatment for Phelan-McDermid syndrome?
A. Yes. If you fast-forward to the end of the presentation, you'll see the acknowledgments, which includes the whole team.
Q. Yep. Okay. Let's go to IGF-1. That's insulin-like growth factor 1, right?
A. Yes.
Q. It's a hormone that's similar in molecular structure to insulin, right?
A. Similar, yeah.
Q. It's produced primarily by the liver?
A. Correct.
Q. It's characterized by the association of intrauterine growth

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retardation with intellectual deficit, right?
A. Sorry, can you repeat that?
Q. It's characterized by the association of intrauterine growth retardation with intellectual deficit?
A. That's taken out of context. It is a growth factor that's critical for brain development.
Q. Okay. It's caused by a deregulated lipid metabolism?
A. What's caused by a deregulated lipid metabolism?
Q. IGF-1 syndrome?
A. Ah. You are talking about a short stature syndrome.
Q. Is that right?
A. So there is a syndrome that's characterized by IGF-1 deficiency.
Q. And is placental dysfunction a cause of IGF-1 deficiency?
A. So we're talking about an indication for IGF-1 as a compound. That's not at all what I was using it

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for. I was using it for Phelan-McDermid syndrome.
And I'm using it to take advantage of the mechanism which is to produce growth.
Q. I understand. But my question is, is placental dysfunction a cause of IGF-1 deficiency?
A. I'm not an expert in IGF-1 deficiency.
Q. Okay. During gestation, a placenta is one of the major sources of IGF-1, right?
MS. BROWN: Object to the form.
THE WITNESS: I'm not an expert in IGF-1 deficiency.
BY MR. WATTS:
Q. Okay. Does DNA methylation play a critical role in placental development?
A. I'm not an expert in placental development or IGF-1 deficiency.

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Q. So I won't be hearing you talk about any of those issues at trial?
A. I mean, depends on what I'm asked about IGF-1.
Q. Well, I just asked you. So do you have any answers?
MS. BROWN: Well --
THE WITNESS: I will not be providing any testimony about placental insufficiency or IGF-1 deficiency.
BY MR. WATTS:
Q. Let's talk about Angelman syndrome. Do you know what that is?
A. I do.
Q. Ubiquitin protein ligase E3A, right?
A. That's correct. I'm not an expert in Angelman syndrome --
Q. Do you know what causes it?
A. I do know what causes it. I'm not an expert in this area.
Q. What about 15q duplications?
A. I'm also aware of 15q

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duplication. I'm not an expert in this area.
Q. Okay. Are you an expert in the etiology of that?
A. I'm not an expert in the area of 15q duplications.
Q. Do you know enough about it to know that the EEG signature is almost identical to the beta oscillations induced by benzodiazapine drugs?
MS. BROWN: I object as lacking foundation.
THE WITNESS: I know that there are EEG signatures that are different than typically developing controls in both Angelman syndrome and 15q11 duplication syndrome.
BY MR. WATTS:
Q. Okay. Do you know that the beta oscillations are similar or almost identical to those induced by benzodiazapine drugs?
MS. BROWN: Lacks

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foundation. I object.
THE WITNESS: I was not investigating that specific question when I came here.
BY MR. WATTS:
Q. Okay. Rett syndrome, are you familiar with that?
A. I am.
Q. It's not an inherited disorder, is it?
A. It's a spontaneous de novo mutation that's considered to be within the heritability domain. So it's genetic.
Q. But not inherited?
A. Parents do not pass on Rett syndrome to their children, but...
Q. And Rett syndrome results in problems with protein production critical to brain development, right?
A. Yes.
Q. Williams syndrome, do you know what that is?
A. I'm familiar with Williams

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syndrome, yes.
Q. It's most often not inherited, right?
A. I'm not sure if that's correct, no.
Q. No? It results from not having a copy of the 25 to 27 genes on Chromosome Number 7, right?
A. Right. So, again, inherited versus sort of heritable. It's still embedded within the genetics.
Q. I'm not saying it's not in the genetics.
A. Right.
Q. But it's not heritable from the parents.
A. It's not inherited from the parents.
Q. In other words, if you have a kid that presents with the rare mutation, Williams syndrome --
A. The parent did not have it.
Q. There you go. Okay.
Prader-Willi syndrome. You

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familiar with it?
A. Yeah, same.
Q. Not genetic or -- strike that.
Not heritable, right?
A. Entirely, wholly genetic.
Q. I know. And I said it wrong.
My question is, it's not heritable, right?
A. It is heritable.
Q. Okay.
A. It is not inherited.
Q. There you go.
And that's an unstable region that's on Chromosome 15, right?
A. 15, yes.
Q. Okay. Smith-Magenis syndrome?
A. I've seen one case with Smith-Magenis syndrome.
Q. And it was not inherited, was it?
A. It's not inherited. It is

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still genetic.
Q. Okay. I mean, all of these are genetic mutations and, therefore, genetic, right?
A. Correct, just like autism.
Q. Yeah. It's just different kinds of a fractured femur that are -- they are all a broken leg?
MS. BROWN: Objection to the form of the question.
BY MR. WATTS:
Q. All right. Let's get -- let's get back to the details.
DiGeorge syndrome?
A. Yes.
Q. That's 22q11.2 deletion syndrome?
A. Yes.
Q. 90 percent of those cases are not heritable?
A. Again, heritable to me means the percentage of the phenotype that's explained by genetics, in which case 22q13 deletion -- 22q11 deletion system

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is heritable. It is genetic in origin.
The parents don't also have it and, therefore, pass it on to their kids. But it is heritable, and it is genetic.
Q. It's not passed along by the parents?
A. Correct.
Q. Okay. So far, you've identified about 250 rare genes, you said?
A. So I think the field as a whole is about at, yeah, somewhere around 250 genes.
Q. Okay. The lion's share of those are not heritable, in that you don't see them in the parents as well, right?
A. Those are mainly de novo mutations that occur when sperm meets egg, so they are carried, but they are within the gamete.
Q. And each of those 250 rare genetic mutations could have an

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environmental etiology if the child is genetically predisposed, right?
MS. BROWN: Objection. Improper hypothetical. Calls for speculation.
THE WITNESS: I'm not sure I understand the question.
BY MR. WATTS:
Q. Okay. When you say that we've gone from zero to 100 rare variants to 250, and we expect to get to 500 to a thousand, those are just different examples of different kinds of gene mutations that you're able to see through the technology that we've identified, right?
A. I think it's a combination of the technology being better resolution, and also a combination of the analytics methods improving.
Q. Now, in -- did you write a paper in 2017 with the scientist known as Angarita?
A. Yes.

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Q. Let me put this up real quick. Exhibit 453.
(Document marked for identification as Exhibit Kolevzon 453.)
BY MR. WATTS:
Q. And a statement you make on Page 227.
A. Oh, that's the one that I didn't recognize.
TRIAL TECH: 227?
MR. WATTS: 226. I'm sorry.
BY MR. WATTS:
Q. Under "Pathophysiology," did you write that, "10 to 15 percent of the autism spectrum disorder is caused by rare genetic variants"?
A. At the time that may have been correct.
Q. Okay.
I'm going to be a liar. I have one more Chung video I want to ask you about. I'm sorry. I thought I was done. Exhibit 548. And we may have

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already played it.
Do you remember when she said that genes only explain 20 percent of the cases?
MS. BROWN: Objection. Misstates testimony.
THE WITNESS: No.
MR. WATTS: Play -- play it again. It's on December 26, 2018.
(Video played.)
DR. CHUNG: The bottom line is, for any one person that I see in terms of identifying a cause, I can come up with the answer these days about 20 percent of the time. That's the good news/bad news.
(Video playback ended.)
MR. WATTS: Stop there.
THE WITNESS: So that's not what she said.
BY MR. WATTS:
Q. Okay. Tell me what she had meant -- I'm clearly ships passing in the light, and I'm not trying to be -- tell

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me what she said.
A. Yeah. So what she says is that when she sees 100 kids in her clinic, she does genetic testing on all of them. They all have autism, but only 20 percent of the genetic tests comes back with a specific genetic cause.
Q. Okay. I'll take it --
A. It's the yield of the genetic testing.
Q. And -- and then let's go back to 4 -- 568 for a second. It's the PowerPoint.
And if you can go to Slide 10. This is dated April 25th.
See how she says, "Genetic diagnoses in 8 to 10 percent of the families"?
A. Yeah.
Q. Okay. Is that consistent with your experience?
MS. BROWN: Objection to form.
THE WITNESS: So, you know,

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this depends entirely on when this slide was done versus when the video was done versus what the technology's being used.
BY MR. WATTS:
Q. Okay. This is why I'm asking both of them. The video that we just watched saying 20 percent was in December of 2018. This is in a slideshow that's dated April 25th of 2023.
A. So I --
MS. BROWN: I object. Lacks foundation.
THE WITNESS: I don't understand the context of this slide and what exactly she's referring to, what her sample is.
BY MR. WATTS:
Q. Okay.
A. So we need to figure that out first.
Q. Now, here is my question.
And she -- she was at Columbia and is now in Boston, and you

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are at Mount Sinai.
Do you have similar statistics in terms of, for every 100 kids that we run genetic testing on, we only identify a genetic diagnosis 8, 10, 15, 20 percent of the time?
MS. BROWN: Objection to form.
THE WITNESS: So the ascertainment will change the yield for sure.
BY MR. WATTS:
Q. Okay.
A. But I can't answer the question as to whether her ascertainment is different than mine.
Q. Okay. Do you know whether yours is 10 percent versus 20 percent?
A. I know what it is in terms of the general field, where I think we're up to actually 30 percent. I think Dr. Chung would say 30 percent today. And I don't know what this genetic diagnosis kind of --

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Q. Okay.
A. -- cohort is referring to.
Oh, this could be SPARK. Is this SPARK?
Q. I think it is.
A. Oh, okay. So that's a different story altogether.
Q. Now, let's talk about siblings for a second. Why is the rate of autism among siblings 50 times greater than in the general population?
MS. BROWN: Objection to form. Lacks foundation.
THE WITNESS: Because of the common variants.
BY MR. WATTS:
Q. Okay. Which is 50 percent of the genetic contribution, right?
A. So among the genetic causes, it's likely that 50 percent probably can be attributed to common variants.
Q. Now, I've noticed that in all of the autism cases where you've testified, you try to get a genetic test

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done on the little boy or the little girl that's at issue, right?
MS. BROWN: Objection to the form.
THE WITNESS: I usually advise that it's the standard of care. But I don't necessarily inform what happens in terms of their case or their strategy.
BY MR. WATTS:
Q. Okay. And even when the genetic testing doesn't identify a common variant or a rare variant with which you are familiar, you say it's not environmental, it's got to be genetic, right?
MS. BROWN: Objection. Objection to form.
THE WITNESS: So if you want to talk about specific cases, we have to evaluate them specifically. I can't sort of talk about them en masse.
BY MR. WATTS:

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Q. Okay. Well, did the genetic test on the case that you testified in Houston come up negative?
MS. BROWN: Objection to the form of the question.
THE WITNESS: So the question, in Houston -- it wasn't Houston. It was Galveston.
BY MR. WATTS:
Q. Galveston.
A. Galveston. Come on, guys.
Q. Listen, we both moved away from Beaumont.
A. Right.
Q. The case that you testified in 2023 in Galveston -- I said Galveston first --
A. Yeah.
Q. The genetic test came up negative, didn't it?
MS. BROWN: Objection to the form.
THE WITNESS: The question at hand for me in that case was

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whether or not there was a likelihood that child had heavy metal poisoning from baby food and whether heavy metal poisoning had been established as the cause of autism.
BY MR. WATTS:
Q. That wasn't my question.
You did genetic testing on that little boy and it came up negative, didn't it?
MS. BROWN: Object to that question. Lacks foundation.
THE WITNESS: So, I think, two things. One, I didn't do any genetic testing. Two, I don't think the details of that particular case are appropriate to share.
BY MR. WATTS:
Q. Well, I'll tell you what. Assuming I've got his deposition --
A. Mm-hmm.
Q. -- and assuming that I've

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talked to his lawyers who gave me that deposition.
A. Mm-hmm.
Q. None of Ethan's genetic testing showed any genetic abnormalities.
Isn't that true? Didn't you testify to that in your deposition, or your trial testimony, in that case?
MS. BROWN: Well, I'm going to interject, though. To the extent the doctor has concerns that there is a protective order or confidentiality, I don't want him to give any testimony.
Whether you had a conversation with your friends on the plaintiffs' side or not, if he feels that giving testimony about someone's genetic testing would be inappropriate --
MR. WATTS: Yeah, if --
MS. BROWN: -- we have to err on the side of caution there. I'm going to instruct him not to answer that.

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BY MR. WATTS:
Q. Let's err -- let's err on the side that when you testify in a trial, it's a public courtroom and it is a public record, okay? So this isn't something where that nonsense is going to play, okay?
You were in trial in Galveston earlier this year and testified that none of Ethan's genetic testing showed any genetic abnormalities, right?
MS. BROWN: And I'll just give you the same caution. If you are concerned that answering these questions is revealing something that was not testified to in a public arena, then I don't want you to answer it.
THE WITNESS: This is a public record?
BY MR. WATTS:
Q. It is.
A. Then, obviously, what I said

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in that public record, I stand by.
MR. WATTS: Exhibit 513. Page 25.
(Document marked for identification as Exhibit Kolevzon 513.)
BY MR. WATTS:
Q. In the public transcript, in the public testimony, in the public record.
Page 25. There we go.
"And so what we know about Ethan's genetic testing is that none of the tests that he had showed any genetic abnormalities, right, sir?"
The answer was, "Correct," right?
A. To the extent of our knowledge and to the extent of the testing that was done, and where we are sitting today, no identified genetic abnormalities were found.
Q. That was the same as in the Sullens case in 2018. There was no

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genetic test done that was able to detect a genetic cause for this child's autism, right?
A. That is --
MS. BROWN: I object. That's inconsistent with the testimony on the rest of this page.
THE WITNESS: I don't have any memory of that case.
MR. WATTS: Exhibit 480, Page 82.
(Document marked for identification as Exhibit Kolevzon 480.)
BY MR. WATTS:
Q. Lines 19 through 21.
MS. BROWN: And can we take a break when you get to a good spot?
MR. WATTS: Yeah, when I get done with this spot, sure.
BY MR. WATTS:
Q. You see in the Sullens

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versus Walmart case, there was no genetic test done that was able to detect a genetic cause for this child's autism?

A. So in this case -- this is also public record?

MS. BROWN: I don't know. Let's look.

This is a deposition. I don't know. If you have any concerns, don't testify about it.

THE WITNESS: So --

MS. BROWN: Do -- can you represent there is no protective order? I don't know how you got this deposition.

BY MR. WATTS:

Q. Do you have an answer for my question?

MS. BROWN: Well, no, but he's raised a concern, and it's a fair --

MR. WATTS: Alli, you don't represent Walmart. Come on. You're just obstructing --

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MS. BROWN: No, no. But if -- if he's subject to a protective order because he gave a deposition in another case and he's raising a concern about it --

MR. WATTS: I've got the deposition --

MS. BROWN: -- I'm going to advise him not to testify about it.

BY MR. WATTS:

Q. I've got the deposition in the Sullens case. There was a genetic testing done. It showed no genetic abnormalities, again.

MS. BROWN: I'm going to give you the same caution --

THE WITNESS: So, yeah, I think -- if we can dig into these cases, the question at hand here was whether the so-called insult caused this child to have autism. In this case it was crystal clear that it did not.

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MR. WATTS: Objection. Nonresponsive.

MS. BROWN: I object.

BY MR. WATTS:

Q. Was there genetic test done of the Sullens kid and it didn't show a genetic cause for this child's autism?

MS. BROWN: Objection to form.

THE WITNESS: I don't recall the details of this case.

BY MR. WATTS:

Q. What about the Huddleston case, Exhibit 526.

(Document marked for identification as Exhibit Kolevzon 526.)

BY MR. WATTS:

Q. Was there testing done in that case?

A. I don't recall the details of that case.

Q. Doctor, with respect to sibling controls, have you read the

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Solander paper in 2016 that the bias tends to attenuate the estimated effect toward the null in some common scenarios, thus producing a conservative estimate of the true exposure effect?

MS. BROWN: Objection to the form of the question.

THE WITNESS: I would have to see the paper.

BY MR. WATTS:

Q. Sure.

A. I have to see the paper.

Q. Okay. Have you heard that generally, in looking at sibling control studies, that they bias towards the null?

MS. BROWN: Objection to the form.

THE WITNESS: I have heard people make that claim, yes.

BY MR. WATTS:

Q. Do you agree with it?

MS. BROWN: Objection to the form. Calls for speculation.

THE WITNESS: I think --

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yeah, I think it depends on the study, it depends on the design, it depends on the rigor.
BY MR. WATTS:
Q. In what way is a study design with the sibling controls that will bias towards the null?
MS. BROWN: Objection. Overbroad.
THE WITNESS: Can you be more specific?
BY MR. WATTS:
Q. No. You said it depends on how the study is designed. In which way does study design cause a sibling control study to bias towards the null?
MS. BROWN: Objection. Assumes facts. Misstates testimony.
THE WITNESS: So a sibling control study, if you take a mother who took acetaminophen during pregnancy and had a child with autism and you compare the

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rates of those autism offspring to the rates of a parent who didn't take acetaminophen during pregnancy, that would be an effective sibling control that should, ideally, control for some genetic confounding at least.
BY MR. WATTS:
Q. Okay. I want -- one last issue and then we'll break.
An environmental factor, as it relates to autism, really just means anything that's not genetic, right?
MS. BROWN: Objection to form.
THE WITNESS: Yeah, so, broadly speaking, people have included lots of different factors in the environment, some of which could actually be genetic in origin. But by definition, they are not heritable.
BY MR. WATTS:
Q. Okay. Good.

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So when you talk about an environmental factor, we know it's not heritable, right?
MS. BROWN: Objection to the form.
THE WITNESS: So it depends.
BY MR. WATTS:
Q. Now, environmental factors can trigger genetic predisposition, right?
MS. BROWN: Objection. Assumes facts. Lacks foundation.
THE WITNESS: You'll have to give me a specific instance.
BY MR. WATTS:
Q. Well, epigenetics is the study of how various factors can influence the expression of genes without changing the structure of the DNA, right?
A. That's a true statement.
Q. And the expression of autism genes may be influenced by environmental factors, right?
MS. BROWN: Objection to the

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form.
THE WITNESS: So theoretically and hypothetically, that is possible.
BY MR. WATTS:
Q. You cited to the Johnson paper, which I marked as Exhibit 415, that says precisely that. Do you agree with it?
A. Let's go to the reference.
Q. Okay.
MR. WATTS: Exhibit 415.
(Document marked for identification as Exhibit Kolevzon 415.)
BY MR. WATTS:
Q. 1188. Second column under "Environmental Issues."
"However, the expression of the autism genes may be influenced by environmental factors. Although currently under investigation, these factors may represent a 'second hit' phenomenon that primarily occurs during

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fetal brain development. That is, environmental factors may modulate already existing genetic factors responsible for the manifestation of autism spectrum disorders in individual children."

Did I read that right?

A. This is an important hypothesis-generating idea and something that's being actively pursued.

Q. Well, let's look at another study you cited. 416, which is the Moy and Nadler study?

A. Sorry, the what?

(Document marked for identification as Exhibit Kolevzon 416.)

BY MR. WATTS:

Q. Page 4, please.

A. Yep.

MR. WATTS: Page 4.

TRIAL TECH: That is Page 4.

MR. WATTS: First page. I'm sorry. In the abstract -- pull up

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the abstract.

Second sentence. Third sentence, rather -- well, second and third.

By MR. WATTS:

Q. "Etiology is thought to involve complex multigenic interactions and possible environmental contributions. In this review, we focus on the genetic pathways with multiple members represented in autism candidate gene lists. Many of these pathways can also be impinged upon by environmental risk factors associated with the disorder."

Did I read that right?

A. So like in all these studies --

Q. Did I read that right?

MS. BROWN: Well, let him answer, please.

THE WITNESS: You read the words on the page, but I need to contextualize it by saying that all these studies are saying the

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same thing, which is that there are possible environmental factors that are worthy of study but, at the moment, are hypothesis generating.

BY MR. WATTS:

Q. Let's go to 511, which is the LaSalle paper.

"Epigenomic signatures reveal mechanistic clues and predictive markers for autism spectrum disorder." In 2023?

MS. BROWN: 511?

MR. WATTS: Yep.

MS. BROWN: This box doesn't have it. Hang on.

BY MR. WATTS:

Q. It says, "These findings have demonstrated that ASD etiology is decidedly complex with hundreds of genes and interactions with environmental factors."

Is that what LaSalle says?

MS. BROWN: I object. He

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needs a minute to look at LaSalle.

Lacks foundation.

BY MR. WATTS:

Q. Can you see it right at the bottom of Column 1 on the first page?

A. So the first part of the sentence is categorically true, that ASD etiology is decidedly complex. It is true that it involves hundreds of genes.

Q. And interactions?

A. Interacts with environmental factors, at this point, remains speculative.

Q. Okay. But the words on the screen say, "involving hundreds of genes and interactions with environmental factors," does it not?

A. Those are the words.

Q. Okay.

A. But you are taking them out of context.

Q. Okay. Let's go to Page 5 and take it out of context some more.

MS. BROWN: I hope you're

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not being argumentive.
BY MR. WATTS:
Q. Second column at the top. "Together these results suggested a multi-hit intersecting pathway between genetic susceptibility and an environmental exposure observed through shared epigenomic signature."
Is that right?
MS. BROWN: I object. This lacks foundation to random sentences being read.
THE WITNESS: I need to figure out what they are referencing. This is a review paper.
BY MR. WATTS:
Q. Well, Doctor, in your papers, you published a study in 2012 with Sandin. "There's evidence that nonheritable perinatal events and/or environmental exposures are likely to have a significant etiological role," right?

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A. There is no question that there are other nongenetic factors that play a role in autism. As of yet, we have not reliably identified them. And none of the risk factors have reached the level of causation.
Q. Dr. Reichenberg there at Mount Sinai is very bullish on the idea that environmental factors have great importance with respect to autism, right?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: Dr. Reichenberg is the lead of our environmental and epidemiological group at Mount Sinai in the autism center, so, yes, he is --
BY MR. WATTS:
Q. And then --
MS. BROWN: Let him finish, please.
BY MR. WATTS:
Q. I'm sorry, I thought you were done. Go ahead.

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A. He is very passionate about exploring these risk factors. And if he were sitting here today, he would certainly not say that acetaminophen causes autism.
MR. WATTS: Objection --
THE WITNESS: That's speculative on my part.
MR. WATTS: Yeah. Objection. Speculation.
MS. BROWN: That question called for speculation, so the answer was appropriate.
MR. WATTS: Okay, Judge. Let's keep going.
BY MR. WATTS:
Q. You and Reichenberg have participated in the same grants seeking money from the federal government, the NIH, and others, to study environmental factors, right?
A. Correct.
Q. And let me just put up one of those abstracts, Exhibit 452.

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MS. BROWN: Can we take a break?
MR. WATTS: Almost done.
(Document marked for identification as Exhibit Kolevzon 452.)
BY MR. WATTS:
Q. Exhibit 452 is one of the grant abstracts, "Autism and Prenatal Endocrine Disruptors," that's referenced in your CV as one of the grants that you've worked on?
A. Yes.
Q. And part of what's said here is, "Both genetic and environmental factors contribute to autism spectrum disorder, but environmental factors have been understudied. Because environmental factors are potentially modifiable, they should be a research priority."
Is that what y'all said?
A. That's what we said.
MR. WATTS: Let's take that break.

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THE VIDEOGRAPHER: The time right now is 12:43 p.m. We are off the record.

- - -

(Whereupon a luncheon recess was taken.)

- - -

THE VIDEOGRAPHER: The time right now is 1:32 p.m. We're back on the record.

- - -

CONTINUED EXAMINATION

- - -

BY MR. WATTS:

Q. Doctor, your research has included an examination of risk factors for autism spectrum disorder, correct?

A. Yes. I've reviewed some of the literature and I have some ongoing projects that are doing that.

Q. And you have done studies on environmental risks for autism, right?

A. I'm involved in studies that are looking at environmental risks for

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autism, yes.

Q. And having done studies on the environmental risks for autism, it is not your testimony under oath that there are no studies that suggest or find that there are causes of autism other than genetic, correct?

A. Can you repeat the question?

Q. Having conducted or participated in the studies on environmental risk for autism, it is not your testimony under oath that there are no studies that suggest or find that there are causes of autism other than genetic?

A. So there's -- there's sort of a double negative built into that.

My testimony is that the only established causes of autism are genetic in origin. And there are ongoing studies of environmental risk factors.

Q. Let me show you your testimony in the Purdie versus Mercy Medical case, Exhibit 486.

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(Document marked for identification as Exhibit Kolevzon 486.)

BY MR. WATTS:

Q. It's August 4, 2020. Page 155, Lines 15 through 18.

"Question: Is it your testimony under oath that there are no studies that suggest or find that there are causes of autism other than genetic?"

Is your answer: "No, that is not my testimony"?

MS. BROWN: Objection to form.

THE WITNESS: I think I'm probably saying the same thing now as I was saying then. That's what's written on the page, yes.

BY MR. WATTS:

Q. Okay. You wrote a paper with a scientist named Puleo in 2012?

A. You'll have to refresh my memory.

Q. Okay.

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MR. WATTS: Exhibit 429, please.

(Document marked for identification as Exhibit Kolevzon 429.)

BY MR. WATTS:

Q. "Advancing paternal age and simplex autism."

Were you one of the authors together with Puleo in this article, 2011 or '12?

A. Yes, I was.

Q. Okay. If we look at Page 368, part of what you write is, "Causal gene mutations in male sperm cells, environmental exposure with mutagenic effects, increasing use of infertility or assisted reproductive technologies, or the combination of these mechanisms may all be potential descriptors. Such mechanisms may particularly play a role in autism spectrum disorder that appears less likely to be inherited, occurring in

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families with no prior history of the disorder, with spontaneous mutation hypotheses thus far gaining the most research attention and support."

Did I read that correctly?

A. So the words on the page were read correctly.

The intent was to better understand paternal age effects. And the idea is that as men age, their ability to synthesize sperm is more susceptible to copy errors, and so they pass on mutations to children. So it's, therefore, still heritable.

Q. Did I read the words on the page correctly, sir?

MS. BROWN: Asked and answered.

THE WITNESS: So I did say that you read the words on the page correctly, but, I think, taking those words out of context. It's important to understand what the intent was.

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BY MR. WATTS:

Q. Well, the context is the first sentence that I didn't read. "The etiological implications of these paternal age findings remain unclear," right?

A. So, in --

Q. I'll tell you what. Let me strike the question --

A. We just have to go back to the date of the article.

Q. Let me -- let me re-ask a different question that's on my mind.

Do you see on Line 2 where it says, "Environmental exposure with mutagenic effects"?

A. I see those words, yes.

Q. A mutagen is a chemical agent that increases the rate of a genetic mutation by interfering with the function of nucleic acids, right?

A. Yes.

Q. It permanently changes genetic material?

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A. Depends on whether it's permanent, but it can change...

Q. So environmental exposure can change genetic material, right?

A. I think we've established that environmental exposure can change genetic material. I think the issue is that in autism, that has not been established.

Q. Well, except in your book chapter.

A. Which chapter is that?

Q. We'll keep going.

Doctor, what is ID?

A. ID is an acronym that stands for intellectual disability.

Q. Genetic and environmental factors are implicated in intellectual disability, correct?

A. Genetic and environmental factors are implicated.

Q. Okay. Have adverse outcomes in neurodevelopmental function been associated with conventional medications

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used during pregnancy?

MS. BROWN: Objection to the form.

THE WITNESS: It's a very broad statement. It would be helpful for you to be more specific.

BY MR. WATTS:

Q. Sure. Exhibit 463.

(Document marked for identification as Exhibit Kolevzon 463.)

BY MR. WATTS:

Q. You wrote a paper in 2017 with Viktorin. Do you remember that paper?

A. If I take a look at it, I'm sure I'll remember it.

Q. Okay. Exhibit 463.

Are you a co-author with Alexander Viktorin?

A. Yes.

Q. And where does he work?

A. He worked at Mount Sinai at

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the time, I believe.
Q. Okay. Rudolf Uher, where did he work?
A. I don't know, but we could check the affiliations.
Q. Reichenberg worked at Mount Sinai, right?
A. Yes, for sure.
Q. Sven Sandin worked at Mount Sinai, right?
A. Yes, for sure.
Q. So there's at least four of you that worked at Mount Sinai that wrote this article entitled, "Association of Antidepressant Medication Use During Pregnancy With Intellectual Disability in Offspring," right?
A. Yes.
Q. If we go to the second page, the top left. Part of what you all wrote is, "Genetic and environmental factors are implicated in intellectual disability," right?
A. Yep.

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Q. And they include "factors that affect fetal development, such as uncontrolled diabetes and congenital exposures to infectious agents or toxic agents," right?
A. It does say these are implicated. That's true.
Q. Doctor, did you write a paper with a gentleman by the name of Costales in 2018?
A. Yes.
MR. WATTS: Exhibit 465.
(Document marked for identification as Exhibit Kolevzon 465.)
BY MR. WATTS:
Q. This is in Charney and Nestler's book, Neurobiology of Mental Illness, Fifth Edition. And it's Chapter 63. "Neurobiology of Autism Spectrum Disorder and Intellectual Disability, Animal and Human Studies," right?
MR. WATTS: Go to the next

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page. Keep going. 63. Go. Right there.
BY MR. WATTS:
Q. Do you see that, sir?
A. Yes.
Q. And if we could go to the next page, 856. Says, "Nongenetic factors, including advancing parental age, have also started emerging in autism spectrum disorder, although the precise nature of these associations has yet to be determined.
"In addition, environmental factors (e.g., maternal alcohol abuse during gestation, infections, birth complications, and malnutrition) are major contributors of ID etiology."
Is that what you wrote?
A. That's what's written. And, of course, I think we've covered this. There's no debate about whether or not there are environmental factors. The debate is what those factors are.
Q. With respect to those

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factors, there are a group of factors that likely act on the genetic vulnerability to increase the risk of autism spectrum disorder; is that right?
MS. BROWN: Objection to the form of the question. Lacks foundation.
THE WITNESS: It's correct that that is a good idea and a hypothetical mechanism that's worthy of exploration. But it has not been established or commonly accepted in the scientific community.
BY MR. WATTS:
Q. Well, let's go to your blog.
MR. WATTS: Exhibit 474.
(Document marked for identification as Exhibit Kolevzon 474.)
BY MR. WATTS:
Q. In this blog, you shared research on autism diagnosis and explained current treatments for the

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disease, right?
A. Give me a moment, because I don't -- I don't have any memory whatsoever of this blog. I don't recall blogging.
Q. Well, on Page 3 of this exhibit, it says at the top, "There's also a group of factors that likely act on the genetic vulnerability to increase the risk of autism spectrum disorder. These risk factors include very low birth weight, preterm birth, older paternal age, and exposure to several toxins during pregnancy"; is that right?
A. So going back to the page before, it says that autism is primarily a genetic disorder. And that, yes, there are likely some environmental risk factors that act to increase the risk.
Q. Okay. And so the bottom line is, if you're genetically predisposed, the environmental factor may occur and push you over the edge, which then leads to the development of autism

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spectrum disorder, right?
MS. BROWN: I object to the form. It lacks foundation.
THE WITNESS: So the hypothesis is exactly that. Correct.
BY MR. WATTS:
Q. Well, it's more than a hypothesis. It was your testimony in the Purdie versus Mercy Medical case in 2020, right?
MS. BROWN: Objection to form.
THE WITNESS: This is a conceptual framework for how environmental factors act, and as I said, there are no established environmental factors that reach the level of causation.
MR. WATTS: Objection. Nonresponsive.
BY MR. WATTS:
Q. Was that your testimony in the Purdie versus Mercy Medical case?

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MS. BROWN: Objection. Lacks foundation.
THE WITNESS: So you'd need to show me exactly what the testimony is --
BY MR. WATTS:
Q. Exhibit 486, Page 140, Line 21, through 141, Line 5.
(Document marked for identification as Exhibit Kolevzon 486.)
BY MR. WATTS:
Q. "Question: I understand your opinion -- all right. I understand the first part of your opinion as a layperson, which is that if you're genetically predisposed, the environmental factor may occur and push you over the edge which then leads to the development of that condition, correct?"
And what was your answer?
A. So --
MS. BROWN: I object. Let's get him the hardcopy.

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Is this something we've already looked at?
BY MR. WATTS:
Q. Sir, what was your answer?
A. I need to see the line of questioning in order to better understand the context.
MS. BROWN: I'll try to find it for you.
THE WITNESS: But my answer, as it's written on the page, is "Yes."
BY MR. WATTS:
Q. Okay. Do you remember the Mercy Medical case, the Purdie case?
A. I remember participating. I don't remember the details.
Q. Okay. In the book chapter that we started this deposition with, in March of 2022, the Textbook of Autism Spectrum Disorder, second division -- Second Edition, your book chapter says, "There is strong evidence that nonheritable prenatal, perinatal and

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parental events play a role in the etiology of autism spectrum disorder," right?

MS. BROWN: Objection to the form.

THE WITNESS: So this is the book chapter that was written by Ori Kapra. Is that -- that's the one we are talking about?

BY MR. WATTS:

Q. It's the one that's got your name on it that wasn't on your CV.

A. Yeah.

MS. BROWN: Well, can we just have the year?

BY MR. WATTS:

Q. 2022 March.

A. So as I said, autism is 80 percent, maybe more, heritable. But when you look at twin studies, there are not 100 percent concordant, which means that there's some environmental effect that plays a role in the etiology. However, there have been no clear

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established factors that rise to the level of causation.

And as it relates to this case, acetaminophen has been studied as it relates to autism, and there's no evidence that it's even associated.

MR. WATTS: Objection. Nonresponsive.

MS. BROWN: Object.

BY MR. WATTS:

Q. In your book chapter, March of 2022, one of the key points says, "There is strong evidence that nonheritable prenatal, perinatal, and parental events play a role in the etiology of autism spectrum disorder"; is that right?

MS. BROWN: Asked and answered. I object.

THE WITNESS: I would say strong evidence exists for some factors. But acetaminophen is not among them.

BY MR. WATTS:

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Q. The etiology of autism spectrum disorder is multifactorial, includes a combination of genetic and environmental factors as well as their interaction, right?

A. Theoretically, in some cases, that may be right. In most cases, I would identify to date, a single genetic factor is sufficient to cause autism.

Q. In the nontheoretical March of 2021 paper you wrote with Katz, that's been marked as Exhibit 491, you wrote, "The etiology of ASD is thought to be multifactorial and includes a combination of genetic and environmental factors, as well as their interaction," did you not?

A. That's what's written on the page. But in the broader context of autism spectrum disorder, as I said, there are many causes of autism where a single genetic mutation is sufficient to cause a phenotype.

Q. Doctor, I want to talk to

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you about comorbidities. Explain for the jury what a comorbidity is.

A. A comorbidity is a condition that occurs along with another condition. So in the case of autism, a common comorbidity would be something like anxiety or attention problems or hyperactivity.

Q. Okay. Did you write a paper with Vahe Khachadourian of Mount Sinai on comorbidities and autism spectrum disorder and their etiologies?

A. It's vaguely familiar.

Q. It says published online in February 2023, this year. Does that help? Published in Translational Psychiatry?

A. Let's take it out so I can see.

MR. WATTS: Okay. Let's go to Exhibit 514.

(Document marked for identification as Exhibit Kolevzon 514.)

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BY MR. WATTS:
Q. First of all, did you write that paper with Mr. -- or Dr. Khachadourian?
A. Dr. Khachadourian wrote the paper. I was a co-author.
Q. And in terms of the other authors of the paper, Behrang Mahjani at Mount Sinai, right?
A. Yeah. Behrang, Sven, Joe, Avi and Magdalena are all at Mount Sinai.
Q. Everybody on this paper is a Mount Sinai scientist, right?
A. Yes.
Q. And in the paper, if we look at Page 10 of 17, the seven Mount Sinai scientists co-authoring this paper end the discussion by saying, "These results suggest that the higher rates of certain comorbidities in autism spectrum disorder may be partly attributable to the higher rates of the underlying risk factors (environmental exposures, or the underlying genetic variation) among the

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affected individuals, rather than to downstream effects of autism spectrum disorder itself."
Is that what you all wrote?
A. Yeah, I'm not prepared to comment on this paper without looking back at the methods.
Q. Okay. Are those words on the page?
A. Well, you've read the words on the page correctly.
Q. Okay.
A. But in order to interpret them I need more time on this paper.
Q. Sure.
The next page has a Figure 5. This Figure 5 shows lines between genetic factors and environmental exposure, right?
A. Yes.
Q. It has a direct line between environmental exposure to autism spectrum disorder, right?
A. Yes.

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Q. It has a direct line from genetic factors to autism spectrum disorder, right?
A. Yes.
Q. And it has direct lines from genetic factors and environmental exposures with respect to comorbidities, right?
A. This is a very simplistic diagram illustrating the interaction between the environment and genetics, yes.
Q. And it's a very simplistic diagram included in a paper that seven scientists at Mount Sinai, including yourself, wrote this year, right?
A. There's no debate that this is a reasonable framework.
Q. Okay. If we go to Page 13 of 17.
Part of which you all concluded was, "We demonstrated that the common comorbidities in individuals with autism spectrum disorder are often

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associated with pre- and postnatal exposure also linked to autism spectrum disorder."
Did I read that right?
A. Again, if you want to go into the results and the conclusions, we need to look at the methods. And I haven't looked at the methods in a long time.
Q. Now, there was, without pulling out the CV, a substantial part of your CV that talks about all of your grants that you've done in the past and done -- or are doing now, right?
A. Yes.
Q. Okay. And without being pejorative about it, you all will go to third-party financiers and seek funding for research on different topics, right?
MS. BROWN: Object to the form of the question.
THE WITNESS: We have a diverse sort of body of funders that may include third parties, as

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you say.
BY MR. WATTS:
Q. Okay. And before I get to the section dealing with Johnson & Johnson funding some of your work, the government funds some of your work, right?
A. The NIH provides grants to fund some of our work, yes.
Q. And the process is, is that they may put out a, I'm going to call it a call bar, for lack -- we want people willing to research X, and then you can make a grant application and they can decide whether to deploy capital to fund your work on that particular subject, right?
A. It's called a request for applications.
Q. Okay. Let's just look at a couple of those. Exhibit 451.
(Document marked for identification as Exhibit Kolevzon 451.)

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BY MR. WATTS:
Q. This is from the Department of Health and Human Services. It's a research project grant. The funding opportunity title is "Environmental Contributors to Autism Spectrum Disorder," right?
A. That's -- that's the title, yes.
Q. If we look at Page 6 of 19. It says the "NICHD is interested in applications that focus on environmental exposures that occurred prenatally during critical windows of fetal development and that impact early childhood development."
MS. BROWN: Let's give you a second to look at the document and to get to where counsel is.
BY MR. WATTS:
Q. Did I read that right?
MS. BROWN: One second.
THE WITNESS: So there is a huge body of priorities, according to different institutes, and among

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them, yes, the NICHD is interested in environmental risk factors.
BY MR. WATTS:
Q. Okay. And you know what the NICHD is. That would be the National Institute of Child Health and Human Development, right?
A. Yes.
Q. And then down at the bottom, it talks about the NIMH, and that's National Institute of Mental Health, right?
A. Right.
Q. Okay. Up at the top, the National Institute for Child Health and Human Development is seeking applications "that focus on environmental exposures that occurred prenatally during critical windows of fetal development and that impact early child development," right?
A. So I think what this points to is the importance of exploring this area.
Q. Okay.

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A. And there are many, many people that are looking to fund this area, because we need to learn a lot more about it.
Q. Okay.
A. And that's because most of it remains hypothetical.
Q. And about five lines down it says, "Specifically, the National Institute of Child Health and Human Development is interested in studies focusing on prenatal exposures that alter the genetic or epigenetic profile and predispose to autism susceptibility; factors that alter the maternal or offspring microbiome and affect infant development; prenatal exposures to maternal disease, conditions, or medications; and the presence of significant inflammation in utero and how it might be quantitatively related to altered cellular function and development in the offspring."
Did I read that right?

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A. There is an infinite number of factors that are important to study, and it may be relevant.
Q. Did I read that right?
A. The words you read are correct.
Q. And then it says the "National Institute for Child Health and Human Development is also interested in specific gene-environment interactions influenced by prenatal exposures," right?
A. These are important things to study.
Q. Okay. And did Mount Sinai submit a grant application in response to this call for grants?
MS. BROWN: Objection to the form.
THE WITNESS: When was this call posted? These applications were due in 2015, so I can't be certain.
Mount Sinai does investigate various risk factors for autism,

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as you know.
BY MR. WATTS:
Q. Okay. Are you familiar with the Food and Drug Administration fast-tracking testing with respect to people's teeth and hair in order to collect information that can be used to predict autism diagnoses?
A. I am roughly familiar with it. I am not an expert in this area.
Q. Okay. Let me show you Exhibit 496.
(Document marked for identification as Exhibit Kolevzon 496.)
BY MR. WATTS:
Q. It's a news article in Spectrum News by a Laura Dattaro entitled, "FDA Cites Hair-Based Autism Diagnostic Aid As a 'Breakthrough.'"
Do you see that?
MS. BROWN: Give him a minute to read it.
MR. WATTS: Sure.

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THE WITNESS: Okay.
BY MR. WATTS:
Q. On Page 2, the article says, "U.S. Food and Drug Administration, or FDA, has granted 'breakthrough device' designation to a hair-based test designed to aid autism diagnosis."
Do you see that, sir?
A. I do. What they are trying to do is develop a biomarker that will speed the path towards assessment.
Q. Okay. The test, called StrandDx, analyzes the levels of chemicals in a strand of a child's hair to capture a snapshot of her 'exposome'-some of her cumulative environmental exposures and how she regulates certain essential nutrients. The measures suggest how a person's physiology responds to her environment, which can predict her chances of having autism, says Manish Arora."
Dr. Arora is a scientist at Mount Sinai, right?

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A. Yes.
Q. It says, "Previous research from the test's makers suggested that autistic people's teeth contain atypical levels of some metals, and that information can be used to predict autism diagnoses," right?
A. So, taken out of context, what that overlooks is that it wasn't the levels of the metals, it was the way that the metals regulate themselves. And the rhythmicity of the level -- of the metals.
Q. Did I read it right?
MS. BROWN: Let him finish his answer, please.
THE WITNESS: This is a good example of when you read words on a page, the words can be misleading.
And so, yes, you read them right, but they are misleading.
BY MR. WATTS:
Q. Okay. And then give you

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some more context. On Page 3 the article says, "Analyzing hair samples makes it possible to look at chemical exposures and how the body regulates them over time, Arora says, similar to how the rings of a tree can reveal its age and changing environment."

Do you see that, sir?

A. I see that's written. It's generally very broad, and I don't necessarily agree with it.

Q. Now, Dr. Manish Arora is a scientist at Mount Sinai with whom you have published scientific literature, right?

A. Correct.

Q. You've done grants together with him?

A. I don't think that's correct.

Q. How many articles have you published with Dr. Arora?

A. I would have to check.

Q. Okay. Do you find him to be

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a reputable scientist?

MS. BROWN: Objection to the form.

THE WITNESS: I respect Dr. Arora. He is a thoughtful and well-intentioned scientist.

BY MR. WATTS:

Q. Okay. Would you agree that Dr. Arora is a thoughtful and well-intentioned scientist, employed by Mount Sinai, is committed to understanding pre- and postnatal exposures that contribute to autism spectrum disorder?

MS. BROWN: Objection to the form.

THE WITNESS: I think it's important for him to try to understand ways of speeding the way that we get people diagnosed.

I think that these biomarkers in and of themselves are not going to speak much to etiology. But they may help

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people take the early signs more seriously and lead to faster diagnoses.

BY MR. WATTS:

Q. Okay. In trying to develop a biomarker with respect to what we see in a child's teeth or a child's hair, do you agree that that is a biomarker that is trying to look at the chemical exposures that child has been subjected to and how the body is regulating it over time?

MS. BROWN: Objection to the form.

THE WITNESS: So biomarkers can be used for many different things. They --

BY MR. WATTS:

Q. Including environment --

MS. BROWN: Well, let him finish --

BY MR. WATTS:

Q. I'm sorry. I didn't mean to interrupt.

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But including environment, right?

A. So I think some biomarkers can reflect environmental impacts. But those environmental impacts don't necessarily have anything to do with etiology. They just are a marker.

Q. Sure.

A. So what's the cause and what's the effect?

Q. They reflect exposure, right?

MS. BROWN: Objection to the form.

THE WITNESS: They potentially reflect differential exposure.

BY MR. WATTS:

Q. Okay. And if they reflect differential exposure to an environmental agent known to cause or play a role in increasing the risk of ASD that can allow a diagnosis at an earlier point in time and allow for treatment of ASD at an

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earlier point, that's the goal, right?
MS. BROWN: Objection to the form.
THE WITNESS: So that's a serious hypothetical. Because there hasn't been, like, a specific environmental factor that's been established as a cause.
BY MR. WATTS:
Q. So we obviously disagree with each other on that.
But the premise of my question was, that's the goal of the environmental biomarker, through the hair and the teeth, to be able to diagnose something earlier, right?
A. That is one of the goals, yeah.
Q. Okay. Yeah. And along the lines of attempting to understand prenatal and postnatal exposures that contribute to autism, that's a laudatory goal, right?

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A. A laudatory goal?
Q. Yeah.
A. I think we talked about the idea that if you can discover risk factors that are modifiable and reduce the risk, that would be something that's important to do.
Q. Okay. Let's go back to 568, which is Wendy Chung's SPARK PowerPoint. Page 42.
She ends, "We're committing to understanding" -- and the last thing that she's committed to understanding is pre- and postnatal exposure contributing to autism.
Do you see that?
A. I see that, yeah.
Q. That's a laudatory goal, right?
A. You know, we all have the same goal. I think the question is how do we interpret the science as it stands today.
Q. Okay.

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A. I think the point that everyone is still searching in many ways underscores how much we have left to know. Especially as it relates to acetaminophen.
Q. With respect to these laudatory goals, do you believe that the government generally tries to fund research that is important to improving the treatment of autism spectrum disorder?
MS. BROWN: Objection. Overbroad.
THE WITNESS: I can't really comment on the government's motivations or priorities. I'm not involved in those decisions.
BY MR. WATTS:
Q. Let's go to Exhibit 521.
(Document marked for identification as Exhibit Kolevzon 521.)
BY MR. WATTS:
Q. This is dated October 22nd

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of 2019.
And this is a Mount Sinai press release. It says Mount Sinai has been "awarded $25 million to study the environment's influence on people's health throughout their lifetimes."
Do you see that?
MS. BROWN: Let's just give him a minute to look at it, please.
THE WITNESS: Yeah, this is Manish's Exposomic Institute.
BY MR. WATTS:
Q. Okay. You said Manish's expo --
A. Exposome Institute.
Q. Institute?
A. Yeah.
Q. Okay. And just so that we've got it in the record. Exposomic means what?
A. It's kind of a word that I only first heard recently. But as I understand it, it relates to the universe

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of environmental exposures, like the genome or the exposome.

Q. Okay. So exposomic is the way in which environmental exposures affects the genome?

MS. BROWN: Objection. Misstates testimony.

THE WITNESS: That's not what I said, and that's not how I understand it.

BY MR. WATTS:

Q. Okay. When asked during your trial testimony in Galveston earlier this year about Manish Arora securing this $25 million grant, that was to explore environmental factors in autism spectrum disorder, wasn't it?

MS. BROWN: Objection to the form of the question.

THE WITNESS: Can you repeat the question?

BY MR. WATTS:

Q. Sure.

MR. WATTS: Let's put up

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Exhibit 513, Page 24, Line 23, through 25, Line 2.

BY MR. WATTS:

Q. You're on the stand on the afternoon of February 16, 2023, and you are asked about this grant.

(Document marked for identification as Exhibit Kolevzon 513.)

BY MR. WATTS:

Q. Have you talked to Dr. Arora about what he's doing with that $25 million that he achieved a grant for?

A. No, not about the grant. I was aware that he got the grant, as I said. I did not know the grant amount.

My focus with Dr. Arora is very sort of narrow as it relates to the teeth study.

Q. With this grant he was able to hire people to study the link between the pregnancy exposures and autism spectrum disorder, wasn't he?

A. I'm not aware of how he

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spends the money or what he's working on.

Q. Let's go to Exhibit 565.

(Document marked for identification as Exhibit Kolevzon 565.)

MR. WATTS: And just blow up the top, starting with Mount Sinai.

MS. BROWN: Okay. Let's just give him a minute to look at the hardcopy if he wants to.

BY MR. WATTS:

Q. This is a press release dated June 30, 2022, from Mount Sinai, entitled, "Clinical Neuroscience Fellowship Explores Links Between Pregnancy Exposures and Autism Spectrum Disorder."

That's the title, right?

A. Mm-hmm.

Q. And then if we go down to the first paragraph, we can see a photograph of a gentleman that I think you're familiar with, Vahe Khachadourian.

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You co-authored the comorbidities paper about autism spectrum disorder with him, right?

A. Yes.

Q. And he's the -- Mount Sinai's first recipient of National Institute of Mental Health's T32 Postdoctoral Research Fellowship, right?

A. That's what it says.

Q. And the Mount Sinai press release says, "A large body of research suggests that environmental exposures during pregnancy may be associated with autism in offspring," right?

A. Yes. The next sentence says, "But those studies barely scratch the surface of the complex task of understanding the cause of autism spectrum disorder."

Q. Then it mentions his mentor Magdalena Janecka, who is working on that intricate puzzle, right?

A. Yeah, that complex puzzle that we're barely able to scratch the

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surface on.
Q. And if we look at Exhibit 566.
(Document marked for identification as Exhibit Kolevzon 566.)
BY MR. WATTS:
Q. Dr. Janecka is working on the subject of functional epidemiology. And the goal of her research "is to better understand why certain parental and early-life factors are associated with the risk of neurodevelopmental disorders in children," right?
A. I've got to open it up.
MS. BROWN: Let's let him open it up and look at it.
Oh, my goodness. If you guys could see the print on this.
MR. WATTS: Guys, just stay focused on the screen. I realize it's small.
MS. BROWN: Yeah, I understand. But just give him a

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minute. Give him a minute.
MR. WATTS: Yeah, just look at the screen -- I'm giving him a minute, but look at the screen.
MS. BROWN: Well, okay, then, I'm going to object. Because what you have on the screen is selected, highlighted parts of an exhibit that you provided us that you couldn't even read with a magnifying glass.
So just give him a minute --
MR. WATTS: Overruled. Let's go.
I am giving him a minute. I just don't want you talking.
MS. BROWN: And if he wants to look at the rest of it, he'll have to ask for you to scroll down.
MR. WATTS: Look all you want.
MS. BROWN: Presumably, you want truthful and accurate

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testimony.
MR. WATTS: I want him testifying, not you.
MS. BROWN: I'm not testifying.
MR. WATTS: You know you're still my buddy, but you need to kind of let him go.
MS. BROWN: I like you, too. But I can't read this.
MR. WATTS: You're the one that wanted paper. I've got a big screen.
Let's go.
THE WITNESS: Can you repeat the question?
BY MR. WATTS:
Q. Sure.
Dr. Janecka, working under the title "Functional Epidemiology," as part of the functional epidemiology lab of the Seaver Autism Center at the Icahn School of Medicine.
Who is the head of the

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Seaver Autism Center?
A. Joseph Buxbaum.
Q. Okay. Have you ever been the head of it?
A. No.
Q. What is your role at the Seaver Autism Center?
A. I'm the clinical director.
Q. Okay. And is Joseph Buxbaum your boss?
A. Yes, one of many.
Q. And so we've got the functional epidemiology lab at the Seaver Autism Center. And, "The goal of our research is to better understand why certain prenatal and early-life factors are associated with a risk of neurodevelopmental disorders in children," right?
A. So it's very hard for me to comment on sentences that are lifted from a web page.
But if you're asking me if you read the sentences correctly, the

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answer is yes.
Q. Is autism spectrum disorder a neurodevelopmental disorder in children?
A. As defined by the DSM, yes.
Q. Okay. Let's go back to biomarkers for a second. You remember we were talking about teeth and hair to try to get a biomarker showing exposure to environmental agents?
A. So we talked about the studies in teeth and hair to show that there are potentially different levels, or different rhythms between levels, that distinguish between autism and not autism.
Q. Okay. I want to simplify our discussion on what a biomarker is. Now, this is going to surprise you because I'm the picture of cardiovascular health. But every once in a while, I'll get my blood taken to get a blood screen, right. And you can get all sorts of data with respect to what your levels are on

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different things.
Those are biomarkers, right?
A. Those are examples of biomarkers, yes.
Q. So, you know, for example, hemoglobin A1C is a measure in your blood for a risk of diabetes, right?
A. It's a biomarker for diabetes, yes.
Q. Blood pressure for heart disease?
A. Yes.
Q. Cholesterol for heart disease?
A. Those are all examples of biomarkers.
Q. Okay. And so by analogy, those are examples of biomarkers that, if they are too high, are likely to cause an increased risk of heart disease for my example, right?
MS. BROWN: Objection to the incomplete hypothetical.
THE WITNESS: So you are

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representing biomarkers that are linked to the cause of a disease that indicate increased risk. That doesn't necessarily mean that there aren't biomarkers that simply identify cases that have already been established.
BY MR. WATTS:
Q. Okay. Fair enough.
So your research is focused on developing biomarkers, among other things, right?
A. Yes, I'm very interested in biomarkers.
Q. Okay. And I think that one of your prior reports said that you've had approximately $4.3 million in grant funding as a principal investigator on a variety of different things, including discovering biomarkers for ASD, right?
A. Yes.
Q. Okay. And I think you described that effort as, we're trying to think about other factors that kind of

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correspond to autism that we can use to predict autism, right?
A. Yes, I'm happy to talk more about those.
Q. Okay. Now I want to play a video of you at 460.
(Document marked for identification as Exhibit Kolevzon 460.)
MR. WATTS: And this is the Advances in Autism conference in 2017.
(Video played.)
DR. KOLEVZON: You can't just think about autism as one group. It's really important for us to develop and validate biomarkers. I think that is going to be the key to clinical trial success, as I said.
(Video playback ended.)
BY MR. WATTS:
Q. What you said is true, right?

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A. What I'm talking about there, electrophysiological biomarkers that have nothing whatsoever to do with the etiology or cause of autism.
Q. Okay.
A. They are measuring autism and, potentially, autism symptoms and a way of identifying subgroups and predicting treatment response.
Q. Now, I noticed that last night I got a Supplemental Rule 26(a) disclosure. I don't know whether you know what that means, but it says that you looked at more stuff. Okay?
A. Okay.
Q. And I think it listed, you know, various reports, the deposition transcripts for the expert depos that have been taken thus far, and then I think there were three new studies that we'll talk about in a little.
You read Dr. Chung's transcript of the deposition taken on the 30th of August 2023, the rough

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transcript?
A. I read most of it.
Q. Okay. Do you remember the discussion about the slide with the concentric circles of genetics and environment?
A. I do, yes.
Q. Let me put up an example, that Exhibit 568.
This is the SPARK and Future of Autism research, April 25, 2023. And go to Page 5.
And does this comport with what you were reading in the transcript when we were asking about this slide?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: Comport, in a critical piece of the slide, to me, is the implication that genetic factors and environmental factors are roughly equal, which, of course, that's 100 percent not the case, and not consistent with

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her testimony.
BY MR. WATTS:
Q. I realize it wasn't consistent with her testimony after she got hired in this case.
But do you know how many different PowerPoints Dr. Chung has used with the environmental factors circle, the identical side of the genetic factors?
MS. BROWN: Well, I object on a number of grounds, including argumentative, and it lacks foundation. It's false, and it misrepresents the testimony and the document.
BY MR. WATTS:
Q. Go ahead, sir.
A. So I'm not aware of how many PowerPoints and exactly what the size of her circles are.
But I am aware of the article that she's written and the body of literature that suggest very

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consistently that 80 to 90 percent of autism is genetic in origin.
MR. WATTS: Objection. Nonresponsive.
MS. BROWN: Objection.
BY MR. WATTS:
Q. Here's my question. Do you know how many times this slide has been used in her PowerPoints over the last decade?
MS. BROWN: How would he possibly know that? I object.
MR. WATTS: Object to form. Come on.
MS. BROWN: I object to the form.
BY MR. WATTS:
Q. All right. Now let's go on.
You can tell me whether you possibly know that.
A. So the size of the circles are irrelevant. But, no, I am not aware of how many times she's presented on this particular slide.

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Q. So the relevance to me is that every time she gives a PowerPoint, she gives it to an audience.

Does that make sense?

A. That makes sense.

Q. And every time you use a PowerPoint with genetic factors having a circle the same size as environmental factors, you are communicating messaging to your audience that is seeing the PowerPoint?

MS. BROWN: Object. Lacks foundation.

BY MR. WATTS:

Q. Make sense?

A. So the intent behind this slide is to teach a lay audience, and the size of the circles is not what's important. It's what comes out of her mouth that's important.

Q. I mean, part of what we're doing here is we put the cards on the table. Is what comes out of your mouth after you are retained as an expert in

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litigation -- you know, there may be some secondary bias that is alleged on both sides.

You've read all the depositions, right?

A. No.

Q. You haven't read my good friend Mr. Murdica, before you started taking money from plaintiffs' lawyers, blah blah blah. Did you see any of that?

MS. BROWN: Objection to the form.

THE WITNESS: I can tell you there were depositions that I read.

BY MR. WATTS:

Q. Okay. And, then, the point is, is that these PowerPoints were given before anybody was hired by lawyers in this case. Can we agree to that?

MS. BROWN: Objection. Calls for speculation.

THE WITNESS: I think what you're implying is that Dr. Chung

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is adapting her opinions about the genetics of autism based on being paid, and that is factually incorrect, because her opinions are based on literature and based on decades of research and have been consistent since the time that she started in this career.

BY MR. WATTS:

Q. Do you think it's fair for a lawyer like me to probe what people who are experts are saying in litigation versus saying what they said before litigation?

A. Absolutely.

Q. Okay. And so, for example, if you said something in a book chapter before you were hired, that's a legitimate area for me to probe, and then you can give your reasons for that, right?

MS. BROWN: Objection to the form.

THE WITNESS: I think it's

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legitimate for you to probe and ask questions. I just think the questions need to be reasonable.

BY MR. WATTS:

Q. And it's reasonable to say, you know what, I'm looking at a PowerPoint slide that an expert for the defense has used in 2023, in 2019, in 2017, in 2014, and communicated to crowd after crowd after crowd before she was retained as an expert in this case.

That's a reasonable inquiry, don't you think?

A. No.

MS. BROWN: Objection to the form.

BY MR. WATTS:

Q. Okay.

A. I absolutely do not think it's reasonable to infer by the size of circles what the genetic component of autism is.

Q. Sometimes you can't believe your lying eyes, huh?

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MS. BROWN: Objection to the form. It's argumentive.
And our realtime has stopped working. Could we just pause to get it fixed?
MR. WATTS: Sure.
THE WITNESS: I'm sorry. I don't know what -- can you clarify what does it mean?
BY MR. WATTS:
Q. You've never heard "believe your lying eyes"?
A. I think I've heard it. I don't want to assume. Are you calling me a liar?
Q. It's a great phrase down in Texas. Don't take offense.
MS. BROWN: Can we go off for one second and just get this working?
MR. WATTS: Sure. Sure.
THE VIDEOGRAPHER: The time right now is 2:19 p.m. We are off the record.

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(Short break.)
THE VIDEOGRAPHER: The time right now is 2:27 p.m. Back on the record.
(Document marked for identification as Exhibit Kolevzon 504.)
BY MR. WATTS:
Q. Doctor, I want to switch gears with you and show you Exhibit 504. This was your expert report dated June 3rd of 2022 in the Palmquist versus Hain case. Is that true?
A. Yes.
Q. And we can see it's in the Southern District of Texas. This is the so-called Galveston matter that we've been discussing, right?
A. Okay.
Q. And I'd like to take you to Page 32 of that report.
And as we look at this report, Section VI has what's called a "Bradford Hill Causation Analysis"?

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A. Yes.
Q. And as we look at Page 32, Section A deals with temporality; is that right?
A. Yes.
Q. Page 33, Section B deals with "Strength of Association"?
A. Yes.
Q. Page 34, C is "Consistency," right?
A. Yes.
Q. D is "Dose Response," right?
A. Correct.
Q. Page 35, E is "Specificity"?
A. Yes.
Q. Page 35, F is "Biological Plausibility"?
A. Yes.
Q. G is "Coherence"?
A. Yes.
Q. Page 36, H is "Experiment"?
A. Yes.
Q. And I is "Analogy"; is that right?

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A. Those are all the sections, yeah.
Q. Okay. So in those five pages, 32, 33, 34, 35, and 36, you did a Bradford Hill analysis to suggest exposure to heavy metals in baby food could not be causative of autism, right?
A. That's what I did for this case, yes.
Q. Okay. And there's something you said I want to talk to you about, and that is the idea of temporality.
And I -- let me paraphrase. You tell me if I, basically, got it right.
And that is that your understanding of autism is that it's conceptualized in utero, even if it's not evident until later on, right?
A. Correct.
Q. And, therefore, a baby already outside of the uterus ingesting baby food with allegedly too much metals in it failed the test of temporality

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because the conceptualization of autism was already present, right?
A. As it relates to autism, yes.
Q. Okay. Now, let's put that aside for a second. And if we go to your report -- you didn't do a section-by-section Bradford Hill analysis of this particular case; is that right?
A. So my review of the literature in this particular case did not indicate the need for Bradford Hill.
Q. Okay. Fair enough.
So the answer is you didn't do one, right?
A. I used Bradford Hill framework.
Q. But you didn't list them all out and talk about each of them. Is that fair?
A. It was very hard to get past the consistency one.
Q. Okay. Now, I want to talk to you about temporality, just for a

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second, and developmental time tables in utero. Okay?
You've testified -- well, you said in your report, Exhibit 403, that it's generally established that altered neurodevelopment in autism spectrum disorder begins in utero and likely in the first trimester; is that right?
A. Yes.
Q. Does it continue through the second trimester?
MS. BROWN: Objection to form.
THE WITNESS: So the brain is developing throughout all three trimesters. So the initial insult occurs, as I said, when sperm meets egg, but the manifestations of that insult can certainly occur throughout utero.
BY MR. WATTS:
Q. Okay. You cited a paper called Prem. Are you familiar with that?

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A. Off the top of my head, I'm not.
Q. Okay. Let me -- hold on a just second, because I want to be specific about this.
If you look at Page 24 of your report, Exhibit 403.
And you, in Footnote 50 --
A. We're talking about the Payne report.
Q. No, your report in this case.
Exhibit 403. Page 24, Footnote 50.
For the proposition, "Because it's generally established that altered neurodevelopment in ASD begins in utero, and likely in the first trimester, challenges remain in determining which neurobiological features cause ASD versus which are a consequence."
And then Footnote 50 cites to a study by Prem entitled "Dysregulation of Neurite Outgrowth and

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Cell Migration in Autism and Other Neurodevelopmental Disorders"?
A. Mm-hmm. Yeah.
Q. Now, the Prem article I marked as Exhibit 485.
(Document marked for identification as Exhibit Kolevzon 485.)
BY MR. WATTS:
Q. And what I'm going to do -- there's a -- let me just kind of pre-stage what I'm doing. There's a variety of time tables that are listed in Prem. I just want to see if you agree or disagree with those particular time tables.
A. All right. It's been a while since --
Q. I mean, you're welcome to read or I can just take you one by one. Whatever you want me to do --
A. Let me take a look at the abstract.
Q. Yeah. Yeah, that's fine.

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That's where I'm going with these particular time tables.
A. Okay.
Q. Are you ready?
A. I am.
Q. Okay. First question is, in the overview, about three lines from the bottom, it says, "For ASD, more recent genetic studies have also suggested that risk genes largely confer" -- "converge upon the developing human cerebral cortex between Weeks 8 and 24 in utero."
Do you agree with that?
A. So this is not an area of my expertise. This is the pathology.
Q. Okay. Let's go to 110.
The next one.
In the middle of the page it says, "During the 8 to 24-week window of human neurodevelopment, neural precursor cells are actively undergoing proliferation, migration, and early differentiation to form the basic cytoarchitecture of the brain."

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Agree, disagree, or don't know?
A. I generally understand that to be a true statement.
Q. Okay. Go to Page 111.
"The formation of the neural tube occurs during early gestation in humans, 3 to 4 weeks."
Agree or disagree?
A. I don't know the answer to that.
Q. Okay. Page 112, down at the bottom.
"Migration of cortical neurons occurs between E-19 and E-22 in rats, but in humans migration begins at 18 weeks postconception and can continue until Week 36, though most cortical neurons are in place by 24 weeks."
Agree, disagree, don't know?
A. You know, sitting here, I don't know for sure, so I don't want to guess.
Q. Okay. Fair enough.

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126. "Moreover, the importance of very early cortical development was recently confirmed by the large scale, over 35,000 samples, whole genome sequencing study by, Footnote 167, that shows that 80 percent of 102 high-risk autism spectrum disorder genes are expressed in the forebrain by 23 weeks gestation and regulate maturing or mature neurons of both excitatory or inhibitory lineages. Thus, while ASD-risk genes may have different roles in the adult brain, a majority of these risk genes seem to play a key role in the regulation of neurodevelopment."
With respect to the 23 weeks after gestation with ASD genes being expressed in the forebrain, is that something you agree with or you just don't know?
A. I don't know.
Q. Okay. Page 135, down at the bottom, and the top of 136.
It talks about, "More

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recently, with wider use of 'omic' studies and more sophisticated modeling tools, pathway analysis studies of idiopathic and syndrome-associated ASD genes have uncovered their" -- "have uncovered their convergence onto the cerebral cortex of the developing mid-fetal brain (8 to 24 weeks old)."
Do you agree with that?
A. Again, the timing of all of these events, I'm not comfortable testifying to.
Q. Okay. I think the same answer, but let me ask.
Interesting -- "Interestingly, in human brain development, it is believed that the first synapses begin to form between 20 and 24 weeks of gestation and the peak of synapse formation occurs postnatally."
Agree, disagree, or don't know?
A. I can't be certain.
Q. Okay. And then on Page 136.

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"Indeed, in the 8 to 24-week period of brain development, we find that NPCs are proliferating, migrating, and differentiating to form the brain."

Any thoughts?

A. I can't know for sure.

Q. Okay. I want to talk to you about the concept of plausible biological mechanisms.

And if I could, I want to go back to 494, which is the book chapter. It's in the second edition of the Autism Spectrum Disorder.

And you see where it says, "We present plausible biological mechanisms linking the" -- "those risk factors to autism spectrum disorder"?

A. I see where it's written.

Q. And then there's a list of prenatal exposures and maternal conditions, including antidepressants and depression, additional prenatal exposures and maternal -- additional prenatal exposures and maternal conditions.

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Gestational diabetes. Maternal high body mass index. Fetal distress and cesarian delivery. Viral and bacterial infections. Acetaminophen. Metals. Folic acid. And air pollution.

Were those all listed in the book chapter that carries your name as a co-author in 2022?

A. Those were listed, yeah.

Q. And then there's a section entitled "Perinatal Risk Factors."

Low birth weight. Preterm birth. Fetal growth restriction, being short for gestational age.

Were those all listed as prenatal risk factors for autism spectrum disorder?

A. So they've all been listed, but they don't all have plausible biological mechanisms.

Q. Okay. And then it says, "Parental Risk Factors: advanced maternal age, advanced paternal age, potential etiologic mechanisms of advancing

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paternal and maternal age."

Is that right?

A. And those are a couple of good examples where biological mechanisms are plausible.

Q. But they are listed under the presentation of biological plausible mechanisms in the book chapter, right?

A. So the book chapter says that they are going to present some biological mechanisms. And then the book chapter lists a whole bunch of different areas but doesn't necessarily imply that each of those areas has a biological mechanism that's plausible.

Q. Yeah. Except that it follows, "We present plausible biological mechanisms linking those risk factors to autism spectrum disorder," didn't it?

A. It does. But there are not plausible biological mechanisms linking all of those risk factors to autism.

Q. As we go back to the Elmo, all of that happened right after you all

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said, "We present biological plausible mechanisms linking these risk factors to autism spectrum disorder," right?

A. Well, the fact that the two things occur next to each other in a chapter doesn't mean that one explains the other.

Q. Hmmm. Let's go through a few of these and just talk about it for a second.

Folic acid reduces the risk of autism spectrum disorder, right?

A. No.

Q. Let me show you Exhibit 464. This is a newsletter from the Seaver Autism Center where you are the clinical director, right?

A. I am.

(Document marked for identification as Exhibit Kolevzon 464.)

BY MR. WATTS:

Q. "Working with collaborators in Israel, and using data from a national

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healthcare provider, our team examined the correlation between intake of folic acid and multivitamin supplements with the risk of autism spectrum disorder in the offspring."

Did I read that right?

A. Hold on. I've got to get to the actual section.

Yeah, there's a lower-odds ratio of having autism among women who took folic acid.

Q. Okay. And let's look at what was said next.

"Of the 45,300 children in the study, 570, or 1.3 percent, received a diagnosis of autism spectrum disorder. Maternal use of folic acid and multivitamin supplements before pregnancy was associated with a 61 percent lower risk for autism spectrum disorder in their children compared with children of mothers who did not use the supplements."

Did I read that right?

A. You read that right. And

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this is a great example of where, potentially, there's something else that led the mothers to take folic acid in the first place that protected them from having a child with autism.

So this finding, in and of itself, is not sufficient to say folic acid reduces the risk of autism.

Q. Okay. But Mount Sinai says if you take folic acid and multivitamins before pregnancy, you've got a 61 percent of lower risk of autism spectrum disorder in your children, right?

A. No.

MS. BROWN: Objection. That misstates the document.

BY MR. WATTS:

Q. Okay. And then the -- it says, "Maternal use of supplements during pregnancy was also associated with a 73 percent lower risk of autism spectrum disorder," right?

A. The next sentence, which I think is the important one, is that, "The

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results need to be interpreted cautiously, because other factors, such as lifestyle choices, could play a role."

Q. Okay. But let's --

A. More studies should be conducted to validate these findings.

Q. Okay. But let's take it step by step.

First of all, does the Seaver Autism Center newsletter in 2018 say, "Maternal use of supplements during pregnancy was also associated with a 73 percent lower risk for ASD"?

Did I read that right?

A. The Seaver Autism Center newsletter highlights findings from one study.

Q. Yep. And then --

A. -- and shows --

MS. BROWN: Let him finish.

THE WITNESS: -- this result.

BY MR. WATTS:

Q. And that happened in 2018,

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right? That's -- that's when this is dated, up in the upper left-hand corner?

A. Yeah.

Q. And in February of 2018 in the Poston case, did you say taking prenatal vitamins is -- things are globally good because they reduce risk?

A. You've got to be more specific, sorry.

MR. WATTS: Sure. Exhibit 468, Page 83, 7 through 20.

(Document marked for identification as Exhibit Kolevzon 468.)

THE WITNESS: Yeah.

BY MR. WATTS:

Q. Okay.

A. Right. Just like you don't want to smoke during pregnancy. But this isn't necessarily related to autism.

Q. So they -- they -- it doesn't have anything to do with autism?

A. Well, so you want to reduce

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risk factors in general, modifiable risk factors.

Just like you don't want to smoke during pregnancy, you also want to take prenatal vitamins. Those things are globally good because they reduce risk --

Q. But you talk --

A. -- of all kinds of things, including neural tube defects.

Q. But you're talking about studies of people with autism.

Do you see that there?

A. I'm talking, in the first -- the first section, in the second part I'm expanding. It's, like, an obvious thing you want to do.

Q. Well --

A. Right? So on one hand you specifically want to reduce the risk of autism. On the other hand, just like we take prenatal vitamins, you want to reduce the risk of bad outcomes, hard stop.

Q. Does the data show that

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taking folic acid before pregnancy reduces the risk of autism?

A. There are some studies that show that as an association. I don't think it's definitively determined.

Q. By the way, folic acid is Vitamin B, right?

A. Yes.

Q. Okay. There's other prenatal vitamins that are like Vitamin D, right?

A. Correct.

Q. Vitamin D levels are low in cases of autism, right?

A. So there is some association with levels of Vitamin D or higher -- or higher levels of Vitamin D reduce the risk.

Q. So you can supplement Vitamin D to reduce the risk of autism?

MS. BROWN: Objection to the form. Misstates testimony.

THE WITNESS: Yeah. So what you're -- what you're doing is

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you're taking some isolated findings that are important and worth considering, and you're implying that you then want to make a change in your practice, like take Vitamin D.

But we are not at that point, as a field.

BY MR. WATTS:

Q. It's probably a good idea to take Vitamin D if you want to reduce the risk of autism, right?

MS. BROWN: Objection. Asked and answered. Inconsistent.

THE WITNESS: I would never tell a patient that if they took Vitamin D during pregnancy, they are going to reduce their risk of autism.

BY MR. WATTS:

Q. Would you tell a jury in Galveston, Texas, earlier this year, Vitamin D levels are low in autism and you can supplement Vitamin D to reduce

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the risk, that's probably a good idea?

A. Yeah.

Q. Did you say that, under sworn oath, in Galveston this year?

MS. BROWN: Hold on. Hold on. Let him answer.

THE WITNESS: If I said that, it was a theoretical proposition based on an isolated finding.

BY MR. WATTS:

Q. Exhibit 512. Page 109, Lines 13 through 15.

(Document marked for identification as Exhibit Kolevzon 512.)

BY MR. WATTS:

Q. February 16th, morning trial session in Galveston.

Is that what you said?

MS. BROWN: Can you blow up the question too?

THE WITNESS: Yeah, these are all ideas that we have that

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are relevant. But nobody is making that recommendation.
BY MR. WATTS:
Q. But you told a jury that's probably a good idea, right?
MS. BROWN: Objection to form.
THE WITNESS: You're taking this out of context, Counselor.
BY MR. WATTS:
Q. Every time I quote your words back to you, you tell me I'm taking it out of context.
MS. BROWN: Well, that's not true, but that happens to be what you're doing here.
BY MR. WATTS:
Q. I feel so bad.
A. I'm not saying that you pick -- that you do every time. I'm saying in this particular situation, they are talking in general about modifiable risk factors. I'm giving Vitamin D as an example, if they were low.

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Q. What's a modifiable risk --
MS. BROWN: Wait, wait. Lets let him finish.
BY MR. WATTS:
Q. And I'm sorry, I thought you were done.
What's a modifiable risk factor?
A. So this is an example. If, in fact, we had established that -- so what we know, to some extent, or at least there have been some associations with higher levels of Vitamin D during pregnancy, lower the risk of autism. Right.
That doesn't necessarily mean that taking Vitamin D protects you, but if it did mean that, that would be a modifiable risk factor. We'd say we want to make sure that we have your Vitamin D levels high, because we can modify that through diet and supplements, and that would reduce the risk.
This is a conceptual framework. This is an idea.

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Q. Oxytocin.
A. Yes.
Q. That's a natural hormone that stimulates uterine contractions in childbirth and lactation after childbirth, right?
A. Correct. Among other -- many other activities.
MR. WATTS: Play Exhibit 470 for a second.
(Document marked for identification as Exhibit Kolevzon 470.)
MS. BROWN: Can you --
BY MR. WATTS:
Q. This is a video of you on February the 28th of 2018.
(Video played.)
DR. KOLEVZON: But, in the meantime, because of all the interest in oxytocin, Dr. Buxbaum and his group, including Hala Harony-Nicolas, used oxytocin in

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another model systems. So instead of using a mouse model, they created a rat model. Rats have bigger brains. They're a little bit easier to work with, evidently.
And they looked at some of the electrophysiological measures. So this is a measure of what's called long-term potentiation. It's basically a proxy of synaptic plasticity. And they looked at the effect of oxytocin on these SHANK3 rats, and they found that, actually, oxytocin reversed the electrophysiological deficits. And that's a really important marker of nerve cell conduction integrity, and we saw that there was essentially a reversal of the deficit. And that was a very exciting initial finding.
(Video playback ended.)
BY MR. WATTS:

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Q. What is it about oxytocin that achieves a reversal in the deficit? Or do you know?

A. So these are rats that are missing a copy of their SHANK3 gene -- actually, I think they're homozygous, so they are missing both copies. And there are -- there's some evidence of some oxytocin dysregulation, essentially, based on cellular studies.

And, you know, there's an idea that if you try different compounds in the model systems and they work, then that's an easy target for humans.

Q. Okay.

A. So they did the study, and we did a study in humans.

Q. All right. So --

A. And it failed.

Q. -- if we look at Exhibit 405, which is your CV. On Page 4, we were going through your grants, and you had $265,000 grant to be a co-investigator to study the neural

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effects to sustained oxytocin treatment on children with autism, right?

A. I've had many different grants studying oxytocin.

Q. Yeah, including that one?

A. Yeah.

Q. Okay. Let me play 459, which is a video of you at the Advances in Autism Conference 2017, on November 16th of 2017.

(Document marked for identification as Exhibit Kolevzon 459.)

(Video played.)

DR. KOLEVZON: Many of you have had children who have been assessed for ADHD, and you have a continuous performance test where you are kind of pressing buttons on prompts. That's essentially what this is.

So there's a little rat in the chamber. There's these five holes. The holes light up. The

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rat touches the light with their nose, and they get a reward.

And what happens is you kind of increase the speed of that, which is a measure of their attention. And you also don't have a light at all, which is a measure of their inhibitory control, right.

So obviously the kid that -- the kids. The rats with the missing copy or two missing copies of their SHANK3 gene had real deficits in their attention, then you give those rats oxytocin, you rescue those deficits.

So now we've identified two critical domains that are consistent with the human phenotype, and we've rescued both of them with oxytocin.

So of course, what do you do next? You do a clinical trial with humans.

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(Video playback ended.)

BY MR. WATTS:

Q. What are the two critical domains that were consistent with the human phenotype?

A. In the rats?

Q. Mm-hmm.

A. In this study, social recognition memory and I think some sort of attention measure.

I wasn't listening to myself, but that's what I recall.

Q. Probably watching this, saying who is that guy?

MS. BROWN: You were distracted by the hair.

THE WITNESS: It is nostalgic and sad.

BY MR. WATTS:

Q. Lets see if we can go back.

When you say modifiable risk factors, how many modifiable risk factors have you investigated over the years?

A. I can't possibly recall.

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Q. Okay. Let me ask you the next thing in the book chapter that's got your name on it, in March of 2022.

Advanced maternal age. It is true as a matter of data that there's an increased risk for autism spectrum disorder with advancing maternal age, right?

A. Yes.

Q. I think there's been 11 published epidemiological studies before controlling for potential confounders and 7 after you control for the confounders, that have all demonstrated a relationship between advanced maternal age, over the age of 35, and an increased risk of autism spectrum disorder; is that right?

A. I can't attest to exactly the number of studies. But generally it's accepted that advanced maternal age is a risk factor for autism.

Q. Yeah. And for example, in our textbook here -- that's where I got

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those, by the way. The 11 studies and 7 --

A. Mm-hmm.

Q. It says, "Summarizing results across studies suggest that older maternal age of mothers is likely to increase risk of autism spectrum disorder by 50 percent after accounting for potential confounders."

Is that consistent with your recollection?

A. I think that the odds ratio varies depending on the study. So sometimes it's much higher than that. But that's not inconsistent.

Q. And then we go to advanced paternal age, and we see that when dad is over the age of 40, you start to see a statistically significant increase in the risk of the child having autism. And it's more than 50 percent after taking into account potential confounders, right?

A. Yeah, and I think it's

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important to point out that both of these are occurring through genetic mechanisms.

Q. We're going to get to that in a second.

After dad's over the age of 50, the increased risk of autism spectrum disorder in offspring is more than 100 percent after potential confounders are taken into account. Isn't that right?

A. Again, I can't attest to the exact odds ratios. But it's commonly accepted that advanced paternal age and advanced maternal age are significant risk factors for autism.

Q. Okay. And I'll represent to you everything I just said came out of this book, okay?

A. That's the book that I didn't write.

Q. But the one that's got your name on it?

A. It does, but I can't --

Q. By the way, let me just ask

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you. Do you really believe in your heart of hearts that if I put a subpoena on you and on Hollander and your co-authors, that there's not going to be e-mails back and forth to you with respect to this book chapter at all?

A. Oh, there -- there will be.

Q. Okay.

A. Yeah.

Q. Drafts?

A. No drafts.

Q. No redlines, nothing like that?

A. No. If I had seen acetaminophen in a draft, I would have deleted it.

Q. Well, I don't know. You published this before you were asked to look at acetaminophen, didn't -- weren't you?

MS. BROWN: I object to the form of the question.

THE WITNESS: Again, you're implying that I'm biased.

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BY MR. WATTS:
Q. Well, what I'm implying --
MS. BROWN: Let him finish.
BY MR. WATTS:
Q. What I'm implying is that before March of 2022 and every study you'd ever done, the word "acetaminophen" was never once used.
A. Right.
Q. And then the first time that your name ends up on a published article, it has a section about how acetaminophen plays a role in autism spectrum disorder.
MS. BROWN: That doesn't even make sense. I object.
BY MR. WATTS:
Q. Is there any other publication where you talk about acetaminophen, consciously or unconsciously, before you became a lawyer -- I mean before you became an expert witness in a litigation?
MS. BROWN: Object to the form.

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THE WITNESS: I think what you've pointed out is that none of the literature that I've ever published has the word "acetaminophen" in it.
And my point is that if I had gotten a chance to review this chapter, I probably wouldn't have put acetaminophen in this either, regardless of whether I had been working on this case or not.
BY MR. WATTS:
Q. Well, that's not what you suggested in Tillery when you were his guy.
A. What did I --
MS. BROWN: Whoa, whoa, whoa.
BY MR. WATTS:
Q. I'm happy to consult with your Mr. Tillery --
MS. BROWN: Timeout, friends. I object to that question as argumentive.

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MR. WATTS: Well, get ready for it at trial.
THE WITNESS: Right. So I'd like to just address that for a moment, which is --
MS. BROWN: Go ahead. Because there was a question --
MR. WATTS: Let's wait on that for just a second. Because I'm going to ask you about those stuff.
BY MR. WATTS:
Q. Let me just ask you this.
If you think, down on a common-sense level, what is it about the advancing age of mom and dad that increases the risk of genetic disruption in a child?
A. So as women age, they produce a finite number of eggs, and those eggs become more susceptible to damage.
Q. And why is that?
A. Because as we age,

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everything becomes more susceptible to damage.
Q. Just look at me.
A. Exactly.
Q. Okay.
A. Strike that "exactly" part.
MS. BROWN: No, don't strike it.
BY MR. WATTS:
Q. Let me ask you this --
A. Hold on, let me finish.
Q. Yeah, go ahead.
A. In the dads, it's a little bit of a different mechanism, where dads are always making new sperm. But the mechanism to reproduce sperm becomes faulty, again, with age. Like as your cars age, the engine starts to break down.
Q. Okay.
A. And so those are examples of heritable genetic factors, essentially passed on from parent to child, but the parent isn't affected, so it's not,

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strictly speaking, inherited, but it's still embedded within the genetics.
Q. Have you ever heard the phrase "pack-years" in dealing with epidemiology with smoking?
A. Pack-years?
Q. Yeah.
A. No.
Q. I mean, I think the concept is --
A. Oh, yes, no. I have, I have.
Q. -- if you smoke for a little bit, it's not enough to --
A. Yeah, five years, five cigarettes is -- yeah, 25-year pack --
Q. Yeah. So with respect to environmental exposures, the longer one is exposed the greater their exposure will be, all other things being equal?
MS. BROWN: Object to the form. Lacks foundation.
THE WITNESS: It depends.
BY MR. WATTS:

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Q. Sorry?
A. It depends.
Q. Okay. If somebody is 50 years old and is subjected to a steady-state exposure of a chemical, they are going to have greater exposure at 50 than they would at 40, all other things being equal, right?
MS. BROWN: Object. Lacks foundation.
THE WITNESS: Depends.
BY MR. WATTS:
Q. Okay. When asked to explain paternal and maternal age effects, the older you get, there is an increased occurrence of spontaneous genomic alterations, right?
A. That's the idea, yes.
Q. In addition to that, the older parents are, the higher number of premature and low-birth-weight babies surviving this, right?
A. I do think that there is a correlation between older maternal and

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paternal age and preterm birth, yes.
Q. I'm just reading from Chapter 11 of your book chapter in the second Textbook of Autism Spectrum Disorder.
So is it true or not? Is this another one where Kapra wrote something you wouldn't have signed off on if you'd wrote the chapter with your name on it?
MS. BROWN: Objection to the form.
THE WITNESS: So, again, I think that there's a correlation there. So it is a reasonable thing to say.
This is an example where there is actually a mechanism, right.
BY MR. WATTS:
Q. Okay. Low birth weight or fetal malnutrition is a major contributor of intellectual disability, which is commonly associated with autism spectrum

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disorder, right?
A. So I am not prepared to testify about all the different causes of intellectual disability.
Q. Were you prepared to testify it in your report in the Daniels-Feasel case, Page 21 of 94, on November 8, 2018?
A. I may or may not have been. But as I'm sitting here today, I haven't investigated this.
Q. Well, let's see where you -- let's see where you are in November of 2018.
MR. WATTS: Page 21 of 94 of Exhibit 479.
BY MR. WATTS:
Q. Environmental factors --
MR. WATTS: Page 21 -- I'm sorry. Oh, you know what, 21 of 94. Sorry. You're right. Go back up.
Paragraph 7-A.
I think you're on the wrong page, Bud. There we go.

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BY MR. WATTS:
Q. In your expert report in the Daniels-Feasel case, did you write that, "Environmental factors (e.g., maternal alcohol abuse during gestation, infections, birth complications, and malnutrition) are major contributors of intellectual disability which is commonly associated with autism spectrum disorder"?
A. I did write that, yes.
Q. And after you wrote that in 2018, you were aware of studies showing that the estimated prevalence for autism spectrum disorder diagnosis was about five times greater if the baby was of low birth weight, right?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: Can you repeat that?
(Document marked for identification as Exhibit Kolevzon 532.)

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BY MR. WATTS:
Q. Sure.
Let me show you Exhibit 532, which is a study by Pinto-Martin.
Did you read Dr. Powell's deposition?
A. Bits and pieces of it.
Q. Did you hear the classic part where he called Pinto-Martin, Mr. Pinto-Martin?
A. I did.
Q. And Baccarelli, Mrs. Baccarelli?
A. I did.
Q. Okay. Well, assuming it's Mrs. Jennifer Pinto-Martin, she is the first author of "Prevalence of Autism Spectrum Disorder in Adolescents Born Weighing Less Than 2,000 Grams," published November 2011 in Pediatrics.
If we go to Page 16 of 20 --
A. Hold on. Let me just read it.
Q. Okay.

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MR. WATTS: Pull out the conclusions.
MS. BROWN: Well, let's just give him a minute to read the article.
BY MR. WATTS:
Q. Hold up.
Have you seen this before?
A. This is familiar, but I don't remember the details of it.
Q. Okay. We'll go to the conclusions on 16 to 20. It says, "The estimated prevalence of ASD diagnoses in this LBW cohort", I assume that's low birth weight cohort?
A. Yes.
Q. -- "was five times the prevalence reported by the Centers of Disease Control and Prevention for eight-year-olds in the general U.S. population in 2006. The prospective study, using rigorous diagnostic procedures, confirms that the rate of ASD is elevated among low birth

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weight/preterm survivors."
Did I read that right?
A. I think extreme low birth weight is a clear risk factor for autism. And I think that --
Q. Why?
A. Why is it higher than the CDC?
Q. Why is it a risk factor for autism?
A. It's just been shown in studies -- I don't know exactly what the mechanism is.
Q. Okay. So you don't know why it's true, you just know that, statistically, it is true?
A. It's one of the commonly accepted risk factors in the scientific community. I don't think it's a causal factor.
It could be acting by virtue of preterm birth. It could be acting by genetic susceptibility.
But the reason why it's

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different than the CDC studies is because the CDC studies include lots of kids who don't have autism.

But I'm speculating.

Q. So in the book chapter from last year, Exhibit 494, with your name on it, it says, "Low birth weight defined as birth weight below 2500 grams, 5 pounds 8 ounces, is considered to be a marker for newborns at high risk for later neurological, psychiatric, and neuropsychological problems."

Do you agree with that?

A. Broadly speaking, that's true.

Q. Okay. Preterm birth. In the same book chapter you all cite Persson 2020.

Says, "Finding the relative risk of autism spectrum disorder increased weekly as the date of delivery diverged from 40 weeks."

Do you know why that would be true?

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A. I'd have to look at this reference to better understand it.

Q. Anything off the top of your head just make sense as to why ASD would go higher the farther you are away from 40 weeks?

MS. BROWN: Objection to the form.

THE WITNESS: I'm not sure I understand the question.

BY MR. WATTS:

Q. Okay. In your paper with Viktorin. Environmental factors that are implicated as affecting fetal development, such as uncontrolled diabetes, results in an increased risk, right?

A. Again, we'd have to pull the paper. Shall we have that exhibit again?

You're quoting the Viktorin paper?

Q. Let's use the Katz paper for this one. Exhibit 491. Page 7, please.

BY MR. WATTS:

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Q. And I should say the Katz/Kolevzon paper, right? You were a co-author?

A. I was. I think we looked at this one already. Hold on.

Q. By the way, did Ms. Katz tell you that she was going to put your name on this book chapter -- I mean, this paper?

A. Is that a serious question?

Q. Probably halfway so.

By the way, you brought up a good point. You are the last author. Tell me the relationship between the last author and the first, and the people in the middle.

A. So --

Q. Just generally, I mean, I understand there's variations.

A. Yeah. Generally speaking, the first author is the person who wrote it, or maybe the second author is the person who wrote it. The last author tends to be the senior author.

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MS. BROWN: Here it is.

BY MR. WATTS:

Q. What does that mean to be the senior author?

A. Senior author is the person who kind of organizes the effort, mentors the person who writes the first -- usually the first or second author.

Q. The older you get, the more hair that you lose, you kind of get -- get to the back as a senior author in the -- how does that work?

A. So, I mean, Avi and I are both equally senior.

Q. Yeah.

A. It happens to be that Julia was a resident of mine, and so I kind of assumed responsibility for the whole effort. And Avi was more of a guiding consultant.

Q. Okay. Is the first author usually junior to the last author?

A. No. I think it depends on the type of study. I put myself as first

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authors on very -- you know, studies that I think are really important, that I want to make sure that I'm the first author on.

Q. All right. Okay. And tell me what you said about the second author, usually writing it, or something?

A. Well, so sometimes there's a first author who sort of acts as lead, but the second author does the most -- most of the writing.

Q. Okay. Okay.

A. In the case of my Chapter 11, I was asked, just to clarify, whether I wanted to be on the chapter. So it wasn't that Raz put me on the chapter without asking me. He did ask me.

Q. Okay.

A. It's just that I was unaware of the contents of the chapter, and I neglected, as an oversight, to read it carefully.

Q. Okay. Fair enough.

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So to get back to my point. And I think you've just obviated the need for it. If I were to subpoena all the e-mails, you would have been copied on this stuff, but it was an oversight by you, not having seen it. You didn't look at it close enough, is what you're saying.

Is that fair?

MS. BROWN: Objection to form.

BY MR. WATTS:

Q. Go ahead.

A. So now I'm speculating based on memory. But I suspect that there would have been an e-mail saying, hey, do you want to be on this chapter, we're updating the first textbook chapter. And I just said -- probably said sure. And then that was probably it. Or maybe, oh, we submitted it, is that cool. And I was like, oh, yeah, that's great. Figuring that I trust Avi and I trust Raz, and we were updating an existing chapter, and

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they were putting me on as a courtesy. So I said thank you very much.

Q. Okay. Avi being Reichenberg?

A. Yes.

Q. I'm told that he's in poor health right now; is that true?

A. Avi is doing well.

Q. Good. Okay. I'll leave it there. I'm not trying to dig in.

A. Yeah.

Q. I heard that earlier this week, and it kind of bummed me out.

A. You know Avi.

Q. I know of his work. I don't know him personally.

Okay. Let's keep going.

The Katz paper, 491, we talked about the pregestational diabetes. You see the overall increased risk varied from 1.39 to 1.65.

Do you see that?

A. Yeah, I do. But, again, this is important not to take out of

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context. Because I think the question of consistency and strength of association is especially important in this review.

Q. Yeah. And in the review that was done in the book chapter, it talks about diabetes as a risk factor, right?

A. I'm sure there's some of that out there. Although this paper likely reflects a more updated review of the literature than that chapter.

Q. Okay. High maternal BMI. BMI is associated with a higher risk of autism spectrum disorder, right?

A. So, again, there are probably some isolated studies that show that. But they may not be consistent enough. So I need to look at this paper more carefully and then we can talk about the results.

Q. Well, in the Katz paper on Page 7, is part of what you all say, "BMI was associated with a higher risk of ASD irrespective of other insulin-resistant

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conditions."
It's on the screen if you want to see where it is.
A. So I think the findings from this paper reflect that the only significant associated factor among the ones that we examined were preeclampsia, and other ones showed some association but probably were not consistent enough or didn't show strength of association and that we thought were just sort of warranting further study.
Q. Okay. Can high maternal BMI lead to inflammation?
MS. BROWN: Objection to the form. Overbroad.
THE WITNESS: So that's very broad.
What do you mean by inflammation?
BY MR. WATTS:
Q. What do you mean by inflammation?
A. You're asking me about

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inflammation?
Q. Yeah.
A. Inflammation, where, in the brain?
Q. Well, I'm just asking generally, first of all, and then we'll get to what you've said about inflammation.
A. Yeah.
Q. Can high maternal BMI lead to inflammation during fetal development?
A. I don't know. Maybe.
Q. Okay. What about excessive gestational weight gain?
A. There have been some associations. I don't know that it's commonly accepted as a risk factor for autism.
Q. Does preeclampsia -- that's high blood pressure, right?
A. Preeclampsia is high blood pressure during pregnancy. And I think, yes, there have been more studies and more sort of stronger associations there.

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Q. Between preeclampsia and inflammation during fetal development?
A. No. Between preeclampsia and autism.
Q. Okay.
A. So as a risk factor for autism.
Q. Okay. But let me get back to my question if that's fine. I hope you've found it.
But does preeclampsia have a role in causing inflammation during fetal development?
MS. BROWN: Objection to the form.
THE WITNESS: Hypothetically.
BY MR. WATTS:
Q. Okay. Is it hypothetically in a way that you've written that?
A. Let's go and see what I've written and decide.
Q. Let's go on to maternal infection first so I can stay on my word

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and then we'll get to preeclampsia.
A. Okay.
Q. Maternal infection. Does infection lead to inflammation?
A. So it depends.
Q. On what?
A. On the nature of the infection. The location of the infection.
Q. Okay. What is it about infectious agents and prenatal infections that causes an increase in the risk of autism spectrum disorder?
MS. BROWN: Objection to the form.
THE WITNESS: So there's many different possibilities. I don't think any have been established.
BY MR. WATTS:
Q. Well, let me look at Page 191 of the book chapter. And this is under Viral and Bacterial Infections.
MR. WATTS: 494, please.

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Page 191.
BY MR. WATTS:
Q. It says, "A recent meta-analysis demonstrated a statistically significant association of maternal infection/fever with ASD in offspring, odds ratio 1.32, 95th percentile, CI 1.20 to 1.46. Citing Tioleco at 2021.
"Although casualty has not been firmly established, these findings suggest maternal infection during pregnancy confers an increased risk for autism spectrum disorder in offspring."
And my question is, what is it about maternal infection that, as a clinician, makes sense to you as causing increased risk of autism spectrum disorder?
MS. BROWN: Objection to the form of the question.
THE WITNESS: I think I answered this in saying that there are probably many different

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hypothetical reasons why maternal infection might increase the risk.
BY MR. WATTS:
Q. Have you studied why?
A. I have not studied why.
Q. Okay. You just know that it increases the risk?
A. I know that there are some studies that show an association.
Q. Okay. Congenital rubella syndrome. That results with -- from a maternal infection with rubella during pregnancy, right?
A. Yes.
Q. It's pretty well accepted to cause autism in the offspring, right?
A. So based on very small studies, that has been reproduced in the literature, that people consider congenital rubella to cause autism.
Q. Let me show you your testimony in the Purdie versus Mercy Medical case.
MR. WATTS: Exhibit 486,

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Page 157, Lines 3 through 6.
BY MR. WATTS:
Q. That's something you testified to on August 4th of 2020; is that right?
A. That's correct.
Q. Okay. Now, is preeclampsia a serious blood pressure condition?
A. I'm not sure I would be qualified to gauge whether it's serious or not. But preeclampsia is definitely a blood pressure condition.
Q. In Exhibit 491, Page 9, you and Ms. Katz write in your article that, "Preeclampsia contributes to an increase in the circulation of proinflammatory cytokines, such as the IL-6 and CRP, which can impact the development of neurotransmitter pathways in the developing fetus."
True?
A. So this is certainly a compelling hypothetical mechanism.
Q. Okay. And it's compelling

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because you and Ms. Katz wrote that general -- or, "Gestational hypertension and preeclampsia are consistently found to be associated with an increased risk of autism spectrum disorder in offspring," right?
A. So I think the results of Dr. Katz's review suggest that, among the various factors that she looked at, preeclampsia came up more consistently and stronger than most of the others.
Q. And if we go to Page 9, "The most consistent association across studies was observed for preeclampsia and autism spectrum disorder. This is further supported by a sibling analysis suggesting limiting" -- "limited familial confounding factors," right?
A. That's what Dr. Katz has written, yes.
Q. And on Page 10, you all concluded, "Current evidence from large-scale, population-based epidemiological studies support an

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association between preeclampsia and autism spectrum disorder," right?
A. So there is an association that's --
Q. Is that an environmental cause of autism?
A. So you're sort of moving quickly to this idea of cause.
Q. Is it an environmental association?
MS. BROWN: Well, let him -- let him finish.
THE WITNESS: So I said association. You said cause.
BY MR. WATTS:
Q. I'll take association.
Is it a environmentally induced association between preeclampsia and autism spectrum disorder?
MS. BROWN: Objection to the form.
THE WITNESS: So it's a intrauterine environment. I would think that's true, yes.

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BY MR. WATTS:
Q. Okay. What is it about maternal smoking that leads to an increased risk of autism spectrum disorder?
MS. BROWN: Objection to the form. Lacks foundation.
THE WITNESS: So I think there's probably many different factors that one could theorize.
BY MR. WATTS:
Q. Go ahead.
A. So oxygen deprivation, for one.
Q. What does oxygen deprivation mean?
A. It means the tissues in the body are not receiving enough oxygen.
Q. And why would that lead to autism spectrum disorder?
A. I didn't say it would lead to autism spectrum disorder.
Q. What is it about oxygen deprivation in the tissues that makes

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sense to you as playing a role?
A. I don't think a role has been established. And I don't think that we know what the mechanism is.
Q. Is it mechanism of action? What are you comfortable saying about it?
MS. BROWN: Objection to the form of the question. Calls for speculation.
THE WITNESS: What am I comfortable saying about what exactly?
BY MR. WATTS:
Q. Well, I asked you why maternal smoking increases the risk of ASD. You said oxygen deprivation in the tissues.
MS. BROWN: I object -- object to that.
BY MR. WATTS:
Q. And I'm asking what is it about oxygen deprivation that makes sense to you, as a clinician, could play a role in ASD?

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A. Yeah, so --
MS. BROWN: Objection. Lacks foundation.
Go ahead.
THE WITNESS: So -- yeah, I think you're mischaracterizing what I said.
What I said is there are probably many different potential reasons, hypothetically, why smoking could be associated with autism. And none of those scenarios would somebody say that smoking causes autism.
But among them, if I'm speculating, oxygen deprivation is, you know, probably not very good for brain tissue and so can lead to global problems, hypoxia-related problems, that theoretically, hypothetically, could be related to an increased risk of autism.
BY MR. WATTS:

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Q. You mentioned hypoxia. What does that mean?
A. Just low oxygen.
Q. Okay. In the same way oxygen deprivation, you said, could play a role, hypoxia could play a role?
MS. BROWN: Objection. Calls for speculation.
THE WITNESS: I'm sort of -- I'm using those things kind of synonymously.
BY MR. WATTS:
Q. Kind of as an analogy?
A. No. As synonyms.
Q. Okay. What other things can cause oxygen deprivation?
A. I don't want to speculate.
MS. BROWN: Objection. Calls for speculation.
BY MR. WATTS:
Q. How does oxygen deprivation differ from oxidative stress?
MS. BROWN: Objection. Lacks foundation.

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THE WITNESS: So they are different concepts.
BY MR. WATTS:
Q. What is oxidative stress?
A. So it's lots of different things. I'm definitely not an expert in oxidative stress. I'm not sure I'm comfortable with providing testimony about that.
Q. What is your understanding of what oxidative stress means?
MS. BROWN: Objection. Calls for speculation.
THE WITNESS: Well, as it relates to autism spectrum disorder, my understanding is that there's no consensus in the scientific community that oxidative stress causes autism.
BY MR. WATTS:
Q. And I'm not sure that answered my question, respectfully.
What -- what is your understanding of what oxidative stress

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means, was my question?
A. Well, so oxidative stress is a relatively nonspecific term that relates to reactive oxygen, sort of radicals that are circulating in the system and potentially causing damage to cells.
Q. What way are they theorized to cause damage to cells?
A. There's many, many mechanisms, as I understand it.
Q. Give me the mechanisms that you understand.
A. I don't understand them, necessarily, because I'm not an expert in this area.
Q. Okay. Can I take the last two minutes to say you're not going to testify about oxidative stress one way or the other?
A. So I'm comfortable testifying that, based on the scientific literature, my knowledge as an expert in autism, that it's not commonly accepted

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that oxidative stress causes autism.
But, no, I'm not going to testify about the mechanisms of oxidative stress.
Q. Have you written about oxidative stress as it relates to autism?
A. I don't think I've studied that, certainly not on a cellular level.
Q. Do you agree that oxidative stress may have lasting consequences for offspring health and development?
MS. BROWN: Objection to the form. Lacks foundation. Broad.
THE WITNESS: I think that's a broad statement. If you showed me the context, I could sort of agree or disagree.
BY MR. WATTS:
Q. Sure.
Page 190 of your book chapter published in March of last year. Exhibit 494, Page 190.
MS. BROWN: Can he have your book? Our hardcopy doesn't go

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past the cover page.
BY MR. WATTS:
Q. Do you see the second line under "Gestational Diabetes"?
Do you agree with that statement, "Oxidative stress due to gestational diabetes may have lasting consequences for offspring health and development"?
A. Yeah, I think that's a nonspecific and very vague statement that's kind of hard to say I don't agree with. Because, sure, it's possible, but under certain circumstances.
Q. Okay. Maternal alcohol use is associated with a higher risk of autism spectrum disorder?
A. I don't think that's been well established.
Q. Is maternal alcohol use a major contributor of intellectual disability which is commonly associated with autism spectrum disorder?
MS. BROWN: Objection to the

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form of the question.
THE WITNESS: Maternal alcohol abuse causes fetal alcohol syndrome which causes intellectual disability.
BY MR. WATTS:
Q. What is fetal alcohol syndrome?
A. It's the effects on a fetus when the mother drinks alcohol.
Q. And what is the effect on the fetus when a mother drinks alcohol?
A. I don't know the details. I'm not a fetal alcohol syndrome expert.
Q. You don't know why fetal alcohol syndrome has an effect on a fetus?
MS. BROWN: Objection to the form. Asked and answered.
THE WITNESS: I'd have to be speculating, and I don't want to do that.
BY MR. WATTS:
Q. Okay. Air pollution. Go to

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Page 192, the book chapter marked as Exhibit 494.
Down at the bottom it says, "A recent review of 20 articles published in England from 1977 to 2020 found a strong association between maternal exposure to particulate matter, mostly during pregnancy, and the risk of autism spectrum disorder."
Is that part of what's written in the book chapter that has your name on it as a co-author?
A. Yeah, it's written. But I have no way to evaluate the accuracy of that statement.
Q. Okay. Let's talk about metals for a second. Page 191 and 192.
Now, my friends at Arnold & Itkin showed me the CV that you produced in that case three months after this was published. It didn't have this book chapter on it there either. Are you aware of that?
MS. BROWN: Objection to the

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form. Lacks foundation.
THE WITNESS: So I think I've been very clear. I was unaware of this chapter entirely, which is why it didn't appear on my CV. So there was no selectivity around including it or not including it. It never appeared on my CV until defense attorneys pointed it out to me.
BY MR. WATTS:
Q. Yeah, a month ago.
A. Three weeks?
Q. Have you revised your CV to add it?
A. I haven't yet, but I will for sure.
Q. This is the last time you want to be crossed about this missing chapter?
MS. BROWN: Objection to the form.
THE WITNESS: No. Honestly, I think it's -- to pull out

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sentences from a chapter I didn't write in a non-peer-reviewed journal, when you talk about reasonableness, this is not reasonable.

BY MR. WATTS:

Q. Well, it's a -- it's a book, right?

A. It's a chapter that's outdated by the time it's published, that very few people in the scientific community actually read.

Q. Are you going to participate in a third edition of Autism Spectrum Disorders to clarify things?

A. That's a very good question. I think if I have the opportunity to revise this chapter, I will.

Q. Okay. In the three weeks since these defense lawyers pointed out that this book chapter with your name on it was published in March of 2022, have you had any discussion with Mr. Hollander about doing a third edition so you can

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clarify your position?

A. I have not spoken to Dr. Hollander about clarifying my position.

Q. Okay. If we look at Page 192, part of what's written in this book chapter about metals is, "These findings suggest that metal toxicant uptake and essential element deficiency during specific developmental windows increases autism spectrum disease risk and severity, supporting the hypothesis of systemic elemental dysregulation in autism spectrum disorder."

Is that what it says?

A. I think Manish has a lot of interesting hypotheses around the roles of heavy metals. And that's what he's written, or that's what's being referenced here, yes.

Q. Okay. If we could, let's go to the concept of medications as an environmental risk for autism spectrum disorder.

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If we could pull out Exhibit 556. I want to talk to you about a May 2010 publication from a college known as Harvard.

(Document marked for identification as Exhibit Kolevzon 556.)

BY MR. WATTS:

Q. Did you apply to go to Harvard?

A. Sorry?

Q. Huh?

A. Sorry, say that again.

Q. Did you apply to go to Harvard?

A. I don't think I was qualified to apply to go to Harvard.

Q. I laughed at one of your prior depositions that you said you went to school in Israel, because I didn't get into school here, or something like that. I can't remember.

Where did you apply that you didn't get into such that you went to

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Israel?

A. I only applied in New York, so Mount Sinai, and Cornell, I was waitlisted, and it didn't work out.

Q. Okay. By the way, where did you go to college in Israel?

A. No, I didn't. I went to -- I went to college in the University of Wisconsin.

Q. You said you went to Israel because you didn't get in somewhere. What did you do in Israel?

A. Right. So I went to college in the University of Wisconsin. And then I didn't do very well in my first couple years. So I created some obstacles to get into medical school. And then I was waitlisted in medical schools in New York. So I went to Israel for medical school. Tel Aviv University.

Q. Okay. Were you overexposed to environmental toxicants in your first couple years in college?

A. That is an excellent

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question, Counselor.
MS. BROWN: Objection to the form of the question.
THE WITNESS: Isn't there -- isn't there something where you plead the Fifth or...
BY MR. WATTS:
Q. Designate this highly confidential. I'm just teasing you.
Okay. Let's go to 556.
You kind of served it up on a platter. I had to go there.
A. Good probe.
Q. The -- 556 is a Working Paper Number 10 published by the Center on Developing Child at Harvard University, the National Scientific Counsel on the Developing Child, entitled, "Early Experiences Can Alter Gene Expression and Affect Long-Term Development."
And if you could, I want to be fair and -- why don't you take a look through this real quick and then I'll ask

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you a couple questions about it.
MS. BROWN: And, Counsel, for the record, is there a date on this?
THE WITNESS: 2010.
MR. WATTS: May of 2010, I believe.
THE WITNESS: Okay.
BY MR. WATTS:
Q. Okay. Let's go to Page 1, which it says, "The Issue." And on the right side, it says, "Nutritional status, exposure to toxins and drugs, and the experiences of interacting with varied environment can all modify an individual's epigenome."
Do you see that, sir?
A. I see that. Yeah, this is the beginning of epigenetics.
Q. Okay. Do you agree with that statement?
A. So this is a very broad statement. And I think that there are some cases in which this is true and

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other cases where it may not be true.
Q. Okay. Let's go to the next page, Page 2. The red area first.
"Early prenatal and postnatal experiences and exposures influence long-term outcomes by chemically altering the structure of the genes."
Do you agree with that statement?
A. I think that, theoretically, exposures can influence the structure of genes, as we know, based on epigenetics. I don't think it's necessarily established in autism.
Q. But generally you agree that this is -- this is true, right?
A. So I definitely agree that the field of epigenetics is onto something.
Q. Is on what?
A. Is onto something.
Q. Okay. Good. Let's go to the second column.

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"We are also learning from new scientific discoveries in both animals and humans that environmental factors such as certain drugs or the nutritional status of the mother have the potential to cause epigenetic changes to genes in eggs or sperm cells in the fetus."
Do you agree?
A. Again, this is a very broad statement, and I think in some cases, it's likely true. In other cases, it's probably irrelevant.
Q. Okay.
MR. WATTS: Next page. Blow up the graphic.
BY MR. WATTS:
Q. "How early experiences alter gene expression and shape development."
And Number 1 says, "External experiences, e.g., stress, nutrition, toxins, spark signals between neurons.
"Neuro signals launch production of gene regulatory proteins

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inside the cell.
"Gene regulatory proteins attract or repel enzymes that add or remove epigenetic markers.
"Epigenetic markers control where and how much protein is made by a gene, effectively turning the gene on or off, thereby shaping how the brain and bodies develop."
Do you see that, 1, 2, 3, 4?
A. I do. This reflects the kind of framework of our understanding about epigenetics.
Q. Okay. Do you agree with Concept Number 1 with respect to external experiences?
MS. BROWN: Objection. Broad.
THE WITNESS: So this is a broad statement. And I think it depends.
So there are some cases where I suspect it's true. But in the case of autism, it's not

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established.
BY MR. WATTS:
Q. So -- I'm interested. We have a deposition protocol that says she can say, "Objection. Form." And she says, "Objection. Broad."
And you say this is a broad statement.
MS. BROWN: Must be broad.
MR. WATTS: Must be coached.
BY MR. WATTS:
Q. Let me ask you this.
With respect to Number 1, external experiences, stress, nutrition, toxins spark signals between neurons.
Is that true?
MS. BROWN: Objection to the form of the question. Broad.
MR. WATTS: Well done.
THE WITNESS: So just to respond to what you said. I think when you ask a very broad question, it's hard to answer it yes or no.

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And, I mean, I agree she uses the word "broad," and then I use the word "broad," so it may sound suspicious to you, but I'm also capable of evaluating a question and determining whether or not it's broad. In this particular case, this is a very broad question.
So I think in some cases, toxins can spark signals between neurons.
BY MR. WATTS:
Q. So you, as a scientist, are capable, in responding to my question, telling me it's broad without having to be coached by a lawyer?
A. Yes.
MS. BROWN: I object to the suggestion that there's any coaching going on here.
BY MR. WATTS:
Q. Lets go to Number 2, neural signal --

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MR. WATTS: Maybe that's why we have the limitation "objection to form." Just curious.
BY MR. WATTS:
Q. Number 2, neural signals --
MS. BROWN: Or to make sure people ask the right questions.
BY MR. WATTS:
Q. -- launch production of gene regulatory proteins -- let me try -- she's talking over me again.
Number 2, neural signals launch production of gene regulatory proteins inside cell.
Do you agree that that's true?
A. Yes.
MS. BROWN: I object to the form of that question.
BY MR. WATTS:
Q. Number 3, gene regulatory proteins attract or repel enzymes that add or remove epigenetic markers.
Do you agree that that's

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true?
MS. BROWN: Same objection.
THE WITNESS: There are cases in which that is true.
Again, that's a broad statement.
BY MR. WATTS:
Q. Epigenetic markers control where and how much protein is made by a gene, effectively turning a gene on or off, thereby shaping how brains and bodies develop.
Is that true?
A. There --
MS. BROWN: Same objection.
THE WITNESS: There are circumstances in which that is definitely true.
BY MR. WATTS:
Q. Okay. Let's go to Page 4.
"In addition to adverse experiences, a wide variety of chemicals, nutrients, and drugs are also capable of modifying the epigenome for long-lasting

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effects on gene expression."
Is that true?
MS. BROWN: I object to that question.
THE WITNESS: Potentially, in some cases, that might be true.
BY MR. WATTS:
Q. And number -- Page 5.
"Injurious experiences such as malnutrition, exposure to chemical toxins or drugs, and toxic stress before birth or in early childhood are not 'forgotten' but rather are built into the architecture of the developing brain through epigenome."
Is that true?
MS. BROWN: Objection.
THE WITNESS: In some cases it may be true.
BY MR. WATTS:
Q. Let's go to Page 7. The first sentence in red. Page 7.
"Epigenetic changes caused by the exposure of pregnant women,

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infants, and toddlers to environmental toxins, prescription drugs, alcohol, and illicit substances require an urgent look at what safeguards can be implemented to prevent such exposures."
Do you agree with that statement?
MS. BROWN: Objection to the form.
THE WITNESS: Again, this is a broad statement, and I think if epigenetic changes were demonstrated to have clear negative effects, then I would agree with it.
BY MR. WATTS:
Q. Okay. Further down in the red. It says, "The serious" -- keep going.
"The serious and continuing impact of prenatal exposure to alcohol and a wide variety of chemical substances, including prescription drugs, on child health and development calls for

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a more vigorous approach to environmental policies and public education."
Do you conceptually agree with that statement?
A. I'm not sure that I'm qualified to, like, make regulatory comments.
Q. All right. Doctor, do you agree that intrauterine exposure to divalproex sodium is a predictive risk factor for autism spectrum disorder, right?
A. I think it's been established that Depakote, or valproic sodium, divalproex sodium, increases the risk of autism, yes.
Q. Now, in 2013, in the book "Neuroscience of Autism Spectrum Disorder," Chapter 1.6, Page 97, this is Exhibit 431, Page 97.
(Document marked for identification as Exhibit Kolevzon 431.)
BY MR. WATTS:

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Q. Your book chapter ends with the conclusion, "Improved understanding of the nature and likelihood of medication side effects in autism spectrum disorder may help identify risk factors to predict in advance which individuals are most vulnerable."

Is that what you wrote in 2013?

A. Again, I certainly wrote that. But we have to kind of pull out and see the larger context in what I was talking about here. I don't remember this chapter specifically.

Q. I'm curious. The Katz/Kolevzon study, Exhibit 491. On Page 3 when you all did this analysis, on Page 3, did you exclude studies focused on the use of medications?

A. I think we were focused on metabolic factors in our search criteria.

Q. And so you excluded studies focused on the use of medications, right?

MS. BROWN: Objection.

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Misstates testimony.

THE WITNESS: I think we were focused on metabolic factors, and we didn't specifically include medications. There are many other papers that have looked at medication effects in autism.

BY MR. WATTS:

Q. There we go.

Does this state that you excluded studies that focused on exposures outside the scope of this review, such as use of medications?

A. So I think my testimony is that we focused on metabolic factors and did not include studies of medications or exposures.

Q. Okay. Let me show you Exhibit 520, which comes from the Mount Sinai website.

(Document marked for identification as Exhibit Kolevzon 520.)

BY MR. WATTS:

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Q. The website at Mount Sinai says, "Certain medicines taken during pregnancy may also lead to autism spectrum disorder in the child."

Do you see that?

MS. BROWN: Hang on. Let's let him take a look at it.

And when we get to a good spot, can we take a break?

MR. WATTS: Sure.

BY MR. WATTS:

Q. Can you see that there on Page 1, about two inches down from autism spectrum disorder, under causes?

A. I see that. I note this is part of the early days when there was concerned about the SSRIs, until the studies were properly controlled for, and didn't realize it was a genetic compounding issue.

Q. The early days.

Go to Page 12 of this document. When was it last updated?

MR. WATTS: Page 11.

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Page 10. Page 9. Page 8. 7.

Well, let's go to the upper right-hand corner.

BY MR. WATTS:

Q. Do you see how I pulled it off of the website on August 22, 2023?

You are the clinical director of the Seaver Autism Center at Mount Sinai, and Mount Sinai's website, as of eight days ago, had this information on it, right?

A. Yes. As the clinical director I'm responsible for the clinical operations, not the social media or website operations.

Q. So this is more recent than the date when the defense lawyers in this case told you about the chapter of the second edition Textbook of Autism Spectrum Disorder, right?

MS. BROWN: Objection to the form.

BY MR. WATTS:

Q. This is fresher than that.

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MS. BROWN: Same objection.
THE WITNESS: So, to be clear, that's a vast body of literature about the associations between SSRIs during pregnancy and the risk of autism spectrum disorder, which have now been well-controlled for, and it's pretty clear that there is no effect. Whatever effect existed, existed because of genetic confounding.
BY MR. WATTS:
Q. So --
A. Whether I said that -- anyway, sorry.
MS. BROWN: No. You should finish.
BY MR. WATTS:
Q. When your employer says A, and you say B, do you have any ability to go and get them to clean up their website so they are consistent with you?
MS. BROWN: Objection to the

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form. Misstates the document.
THE WITNESS: So we update our website periodically. And we correct information or provide new information.
Science is an iterative process. But I don't monitor the website on a very consistent basis.
BY MR. WATTS:
Q. When you say we update it, who is involved in that?
A. So periodically our communications director will say, hey, we're taking a look at this page, look at the content, would you change anything, does anything need to be revised, and we'll make changes.
Q. Have you had any recent discussions with your communications director about what's on the website concerning acetaminophen and autism?
A. No.
Q. Even though you were hired

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in this case back on December 15th of 2022?
A. No. I don't use our website as a source of reliable information. I use the literature and peer-reviewed journals.
Q. Why does Mount Sinai have a website in the first place? Surely they are putting up what they think is reliable information.
A. Well, I think that the information needs to be updated periodically in order to remain reliable.
Q. Okay.
MR. WATTS: Why don't we take that break that she asked for.
MS. BROWN: Thank you.
THE VIDEOGRAPHER: The time right now is 3:45 p.m. We are off the record.
(Short break.)
THE VIDEOGRAPHER: The time right now is 3:59 p.m. We're back

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on the record.
BY MR. WATTS:
Q. Doctor, have you done any work in analyzing the prevalence of autism on the island of Cuba?
A. I can't say that I have.
Q. Do you have any information as to whether that prevalence rate is lower or higher than in the United States?
A. So because autism is a genetic disorder, you would expect prevalence rates to be roughly equal across the population.
Q. Yes.
A. However, it would depend a lot on the methodology used for the prevalence studies.
Q. Now, you understand that acetaminophen, also known as paracetamol, is one of the most widely used analgesic and antipyretic drugs in the world, right?
A. I'm aware of that, yes.

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Q. Can we agree that nearly two-thirds of the women pregnant in the United States use acetaminophen during their pregnancy?

A. I read that that statistic is correct, yeah.

Q. That is a higher rate of use than pregnant women across Europe use acetaminophen during their pregnancy; is that right?

MS. BROWN: Objection to form.

THE WITNESS: I haven't investigated rates across Europe.

BY MR. WATTS:

Q. Is acetaminophen available over the counter in the European Union?

MS. BROWN: Objection to form.

THE WITNESS: I haven't investigated the use of acetaminophen in Europe.

BY MR. WATTS:

Q. Okay. You've testified in

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birth trauma cases, right?

A. Yes.

Q. Do you remember the Davis versus Menges and MidHudson Medical Group case?

A. I do remember that case, yes.

Q. A young boy named Rhyder Davis, who came out blue, according to his mother, suffered shoulder dystocia, fractured humerus during delivery.

Does that ring a bell?

A. Yes.

Q. Children with autism show an increased evidence of perinatal complications, right?

MS. BROWN: Objection to the form.

THE WITNESS: Broadly speaking, there are perinatal complications that have been associated with increased risk of autism.

BY MR. WATTS:

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Q. Okay. You wrote that in your book 23 years ago when you were in medical school, right?

A. I may have.

Q. Okay. Perinatal asphyxia can increase the risk of autism?

A. I think epidemiological studies would suggest that hypoxia-related events increase the risk, yeah.

Q. Inflammatory processes can contribute and increase the risk of autism for a subset of cases. You testified to that, right?

A. So I think it would depend on the inflammatory process. And I think it would be important to clarify that we are not talking about causal factors. We're talking about associated.

Q. Well, did you say, "Inflammatory process may contribute and increase the risk for autism in a subset of cases"?

A. If you're quoting me, then I

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assume I said it.

Q. Okay.

For the record, August 4, 2020, Purdie versus Mercy Medical, Page 13, Lines 4 through 6, Exhibit 486.

In the Katz paper, did you and Ms. Katz write -- Dr. Katz -- "A chronic low-grade inflammatory state is thought to be present in obese mothers, which accompanies the fetus during its intrauterine development"?

A. That is thought to be the case, yes.

Q. What is a maternal prenatal metabolic syndrome?

A. I think metabolic syndrome in general refers to risk of diabetes and being overweight.

Q. And that results in an inflammatory state which can have a significant impact on fetal neurodevelopment, secondary to neuroinflammation, and can affect synaptic plasticity, oxidative stress, as

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well as neurotropic and neuroprotective signaling?
A. Those are all hypothetical mechanisms that can be associated with metabolic syndrome.
Q. And you wrote that on Pages 2 and 3 of the Katz paper just two years ago in March of 2021, right?
A. I can look at the paper now and confirm.
MR. WATTS: Sure.
Exhibit 491, Page 2 and 3, please.
BY MR. WATTS:
Q. There it is on the screen.
Is that what you wrote?
A. That's written in there, yes.
Q. Acetaminophen may interrupt brain development by the induction of oxidative stress leading to neuronal death, right?
A. Can you repeat that?
Q. Acetaminophen may interrupt brain development by induction of

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oxidative stress leading to neuronal death.
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: So I think that that's a theoretical mechanism that some people might propose.
BY MR. WATTS:
Q. Okay. You've seen that proposed in the published literature?
A. I've seen that proposed in the published literature.
Q. You've never published that that is incorrect?
A. I have not published about acetaminophen.
Q. In the book chapter from last year, it says it's also been suggested that acetaminophen increases the risk for ASD by causing neuronal oxidative stress, citing to the Ghanizadeh publication, 2012?
A. So that was written by the

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author of that chapter, yes.
Q. Okay. And you are the second author listed in that book chapter, right?
A. I am. But as I've noted, I didn't have an opportunity to review. And if I did, I probably would have changed some of the things in that chapter.
Q. Okay. What's the relationship between endocannabinoid dysfunction and the risk of autism spectrum disorder?
MS. BROWN: Objection to the form.
THE WITNESS: There is no clear relationship. There are some proposed hypothetical relationships.
BY MR. WATTS:
Q. And what's the biologic plausibility of that? Why does that make sense? Why is it being proposed?
MS. BROWN: I object.

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THE WITNESS: I don't think there's been an established mechanism.
BY MR. WATTS:
Q. You don't know?
A. I don't think there's been an established mechanism that's commonly accepted. I think people might propose, hypothetically, that it relates to glutamatergic cannabinoid regulation or, perhaps, hypothetically, to immune regulation.
Q. Okay. You agreed in your report that glutamatergic activity is critical for the brain's plasticity, correct?
A. That is correct.
Q. Excitatory and inhibitory imbalance of glutamatergic activity plays a role in autism spectrum disorder, right?
MS. BROWN: Objection to the form.
THE WITNESS: There's

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evidence of glutamatergic and GABAergic dysregulation. It's not clear whether it's a causal effect or a consequence.

BY MR. WATTS:

Q. By the way, AM404 may activate cannabinoid receptors in the brain and exert effects on glutamate GABA activity, right?

A. I don't know that that's true.

Q. Well, did you write that that was true in your report?

A. I wrote that that is what people have proposed to be true theoretically.

Q. All right. Have you studied whether or not it is true?

A. I am not a toxicologist. I have not studied that, no.

Q. In the Purdie versus Mercy Medical case, August 4, 2020, you were asked, "Would you agree that environmental factors outside of a

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genetic context can affect the glutamate system in the developing fetus?"

Did you say, "Yes, I'd probably agree with that"?

MS. BROWN: Objection to the form.

THE WITNESS: So if you can put that up so I can look at it in context --

MR. WATTS: Sure. Exhibit 486. Page 83, Lines 9 through 19, please.

MS. BROWN: Can you blow up the whole page so we can see it. Thank you.

BY MR. WATTS:

Q. "What role, if any, does the glutamate system play in the final common pathway of autism spectrum disorder?

"I mean, the glutamate system is implicated in many different cases of autism and the glutamate system is relevant to many different genes that underlie autism.

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"Question: Would you agree that environmental factors outside of genetic context can affect the glutamate system in the developing fetus?

"Answer: Yes, I'd probably agree with that."

Is that what you said?

A. I said that. And I would stand by it.

Q. And exposures to various toxins or medicines that affect the glutamatergic system can affect the glutamatergic function of the fetus, right?

MS. BROWN: Objection to the form.

THE WITNESS: Can you say that again.

BY MR. WATTS:

Q. Sure.

MR. WATTS: Page 85, Lines 11 through 14.

By MR. WATTS:

Q. And you said, "Likely

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exposures to various toxins or medicines that affect the glutamatergic system can impact the glutamatergic function in the fetus"?

A. So what I'm referring here to, toxins, right, we're not talking about acetaminophen, just to be clear.

Q. Right. You said or medicines, though, to be fair.

A. To be fair.

Q. Okay.

Is that a true statement?

A. I think in a hypothetical sense, and we're talking in a very broad hypothetical sense, it is certainly a possibility.

Q. Okay. Prenatal exposure to acetaminophen has been associated in a dose-dependent relationship with increased DNA methylation among children with ADHD.

Agreed?

A. So you are talking about ADHD, and I'm not prepared to provide

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testimony as to ADHD.
Q. Okay.
A. I haven't explored that.
Q. The genes affected by DNA methylation are those enriched in pathways involving oxidative stress and neurological function.
Do you agree?
MS. BROWN: Objection to the form.
THE WITNESS: Again, in general, it's hard for me to comment on isolated sentences. So if you give me the context, I'm happy to look at it.
BY MR. WATTS:
Q. Sure.
In your report did you say, "Among those differentially methylated regions, the region containing the CYP2E1 gene was associated with an increased risk for autism spectrum disorder"?
A. Let's take a look at the report.

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Q. Sure.
MR. WATTS: Exhibit 403, Page 69, Paragraph 124.
BY MR. WATTS:
Q. Do you see that, sir?
A. Paragraph 125?
Q. 124. "Among those differentiated methylated regions, the region containing the CYP2E1 gene was associated with increased risk for autism spectrum disorder."
That's in your report, right?
A. So I'm discussing the results from a study by Zhu and colleagues, where that was what their finding was.
Q. Now, that's at Page 69. If you go back to Page 68 just for a second. You see how in Paragraph 123, you cite to the LaSalle paper in Footnote 201?
A. Mm-hmm.
Q. Yes?
A. Yeah.

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Q. Now J.M. LaSalle is Dr. Janine LaSalle, right?
A. I don't know Dr. LaSalle's first name.
Q. Are you aware that she was an author of a study identifying the CYP2E1 gene as an autism spectrum disorder risk gene?
MS. BROWN: Objection to form. Lacks foundation.
THE WITNESS: So I've looked extensively at the genetic literature, and I have not found evidence that CYP2E1 is an autism risk gene.
BY MR. WATTS:
Q. Have you read her article entitled, "Human Molecular Genetics," in 2019?
A. I'm happy to take a look at it.
Q. Now, with respect to Exhibit 481, which is what you cited to, the LaSalle paper -- no, I'm sorry. I've

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got it butchered.
(Document marked for identification as Exhibit Kolevzon 484.)
BY MR. WATTS:
Q. 484. There's our guy from Australia.
A. Back to them, yeah.
Q. Okay. Have you read the Zhu and -- paper entitled, "Placental DNA Methylation Levels at CYP2E1 and IRS2 are Associated With Child Outcome in a Prospective Autism Study"?
A. I have.
Q. And you see how Janine LaSalle is the, what'd you call it, the last author, the senior author?
A. Yeah.
Q. Okay.
In the abstract, go to the abstract. It says, "DNA methylation acts at the interface of genetic and environmental factors relevant for autism spectrum disorder," right?

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A. That's what it says.
Q. And if we go to Page 23 of 48. Part of the conclusion is, "Both CYP2E1 and IRS2 are related to protein synthesis, cell proliferation, and cell metabolism consistent with previous studies of convergent gene pathways in autism spectrum disorder. These results, therefore, provide evidence that placental methylation levels reflect the intersection of genetic and environmental risk and protective factors that are expected to be useful for early intervention and prevention of autism spectrum disorder."
Is that what it says?
A. That's what it says. But I don't agree with the conclusions based on these data.
Q. Now, when placental methylation levels reflect the intersection of genetic and environmental risk and protective factors, what is your understanding as to what is resulting in

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a higher risk of ASD? Is it higher methylation level at the placental area or lower?
MS. BROWN: Objection to the form of the question.
THE WITNESS: So I'd have to go back to the article.
BY MR. WATTS:
Q. You don't know?
A. I'd have to go back to the article.
Q. Okay.
A. But the idea that CYP2E1 is an autism risk gene, to me, is unfounded. So the fact that there is differential expression does not necessarily mean that there's any etiological role.
Q. So, really, I'm not asking about CYP2E1 yet. I'm asking about placental methylation levels.
A. Sure.
Q. And do you agree that placental methylation levels reflect the intersection of genetic and environmental

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risk and protective factors that are expected to be useful for early intervention and prevention of ASD?
MS. BROWN: Objection to form.
THE WITNESS: So very broad. So --
BY MR. WATTS:
Q. Do you agree with that very broad statement?
A. In general, methylation can affect the expression of different genes. And differential expression can be characteristic of certain disease states but doesn't necessarily reflect any etiological role.
Q. Okay. Now, in your report at Page 73 and 74, you write that it's been postulated -- Paragraph 133.
"It's been postulated that disruptions in prostaglandin signaling during early development can lead to adverse developmental outcomes, including autism spectrum disorder."

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Do you see that?
A. Yeah. I'm summarizing some of what the literature --
Q. Have you done any research yourself with respect to prostaglandin synthesis pathways and signaling?
A. So in my 20-some-odd years of experience going to conferences and talking to colleagues, I have some understanding of what the common pathways are and the generally accepted causal theories. And prostaglandin synthesis is not one of them.
Q. So, respectfully, that wasn't my question. I was asking you about you doing original research.
Have you done any original research yourself with respect to prostaglandin signaling --
A. I have not.
Q. Okay. Do you agree that acetaminophen acts as a cyclooxygenase inhibitor?
A. I think that's one of the

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proposed mechanisms of action.
Q. Do you agree that that's true?
A. I don't have the knowledge or background to determine that.
Q. Okay. Do you agree that cyclooxygenase inhibitors prevent prostaglandin synthesis?
A. I understand that that's been postulated. I have no experience to confirm that.
Q. So no way to rebut it or confirm it, is what you're saying?
A. It's outside of my expertise.
Q. Have you read the Addo paper, Exhibit 482.
(Document marked for identification as Exhibit Kolevzon 482.)
BY MR. WATTS:
Q. Have you seen this before?
MR. WATTS: Put that on the left side and put Exhibit 404 up

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on the right.
BY MR. WATTS:
Q. Exhibit 404, if you go to the second page, is your materials considered list.
A. Mm-hmm.
Q. Do you see between Adams and Aishworiya where Addo would go, it's not there?
A. I see that it's not there. And I am not familiar with this article.
Q. Okay. So you haven't read the Addo paper before coming here today, fair?
MS. BROWN: Objection to form.
THE WITNESS: I don't -- I don't recall.
BY MR. WATTS:
Q. Okay. Let's just look at Page 5 real quick.
A. Can you just give me a second to read what this is about?
Q. Sure.

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And part of what's on Page 5, just so you know where I'm going. It says, "Our data suggests that the prostaglandin synthesis pathway may be disrupted in the placenta related to in utero acetaminophen use."
You can see that, right?
MS. BROWN: Take as long as you need.
BY MR. WATTS:
Q. It's up on the screen.
A. So what you've written -- what you've read is what's written.
Q. Okay.
A. I would probably look more deeply into his methods. I think it speaks to the point that I made before, which is that although there could be methylation or expression changes, doesn't necessarily reflect an etiological mechanism.
Q. Well --
A. So I agree that it's written on the screen, but I can't agree or

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disagree, for that matter --
Q. Yeah, you haven't studied it. I mean, this is the first time you've ever seen it, fair?
A. Fair.
Q. Okay. Now, let's go to endocrine disruption for a second.
You agree that the endocrine system plays a clear role in prenatal brain development, right?
MS. BROWN: Objection to form.
THE WITNESS: Yes.
BY MR. WATTS:
Q. You agree that the endocrine system plays a clear role in prenatal brain development, because disruptions in endocrine function can lead to a wide array of adverse developmental outcomes, agreed?
A. I believe you're reading from my report, and I agree with my report.
Q. Now, for that reason, if we

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go to Exhibit 424, which is Mount Sinai's publication in 2012 where they published a list of the top ten toxic chemicals suspected to cause autism and learning disabilities.

(Document marked for identification as Exhibit Kolevzon 424.)

BY MR. WATTS:

Q. The Center for Environmental Health Center -- or the Children's Environmental Health Center -- go to Page 2 -- put out a list and it says, "CEHC developed a list of ten chemicals found in consumer products that are suspected to contribute to autism and learning disabilities to guide a research strategy to discover potentially preventable environmental causes. The top ten chemicals are..."

Is Number 6 endocrine disruptors?

A. So, if you're -- yes, the Number 6 is listed as endocrine

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disruptors.

And all of these are worthy for exploration. And all of these are important to study. But none of these have been demonstrated to be associated with autism.

Q. Acetaminophen is a well-known endocrine disruptor?

A. I can't say that that's true.

Q. Can you say that it's untrue?

A. As I said, I'm not qualified to analyze the endocrine effects of acetaminophen. I can say that it's not associated with autism, however.

Q. Well, acetaminophen may interfere with maternal and neonatal hormones, e.g., the thyroid related to brain development, agreed?

A. So there may be many different mechanisms. It's not clear to me that any of them are reliably established.

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But what has been pretty reliably established is that there isn't a significant association in epidemiological studies between acetaminophen use during pregnancy and autism as the outcome.

Q. Doctor, does serotonin metabolism dysfunction -- does your book chapter from 2022 say it's one of the few consistent biological explanations leading to autism spectrum disorder?

A. So the role of the serotonin system in autism is one of the more consistent findings, although it remains hypothesis generating.

Q. In the book chapter, at Page 187, says, "These findings suggest prenatal exposure to SSRIs may have a causal role in ASD by operating directly on the developing brain."

A. So, thankfully, over time, because science is an iterative process, we've learned that SSRI use during pregnancy is confounded by indication,

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and actually once you control for genetics, you attenuate the effects. So the effect is more about the mom, not about the SSRIs.

Q. And you said that in litigation involving SSRIs?

A. Have I said that in litigation? I probably put that in a report involving SSRI.

Q. What is BDNF?

A. What does it stand for?

Q. Yeah.

A. Brain-derived neurotrophic factor.

Q. Say again?

A. Brain-derived neurotrophic factor.

Q. Okay. Does acetaminophen exposure impact BDNF in the neonatal brain?

A. I'm not an expert in this area. I couldn't say whether that was true or not. I'm sure it's been proposed.

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Q. What you can say is BDNF is a critical growth factor for brain development and plasticity, right?
A. I've said that, and I think that continues to be the case, yes.
Q. All right. Now, with respect to prostaglandin signaling and the risk for autism spectrum disorder, your report says it's derived from gene expression studies in the cyclooxygenase knockout mice, 219, another animal experiment showing effects of autism -- on autism spectrum disorder-related symptoms, right?
A. So when I review the literature --
Q. I'm sorry?
A. I said when I review the literature and I try to figure out from where this theory was derived, it seemed to be, in part, derived from some animal studies, including cyclooxygenase knockout mice.
Q. Okay. Let me show you a

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couple of videos where you talked about this.
MR. WATTS: Exhibit 456. November 16, 2017, at the Advances in Autism Conference.
TRIAL TECH: I'm sorry, did you say 426?
MR. WATTS: 456.
(Document marked for identification as Exhibit Kolevzon 456.)
(Video played.)
DR. KOLEVZON: So then what do you want to do. Think about our model where you're going to go from mouse to physiology, which we understand, to now thinking about treatment.
(Video playback ended.)
BY MR. WATTS:
Q. And let me show you Exhibit 564, also from 2017, but two months earlier on September 10th.
(Document marked for

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identification as Exhibit Kolevzon 564.)
(Video played.)
DR. KOLEVZON: But so this has been sort of our model in terms of treatment development. This is not a novel model, right. This is something that cancer treatments have been following for a long time.
But for us, again, this kind of window into these single genes provides opportunities to, A, you know, discover an actual gene that we consider to be pathogenic, as Colleen explained. And then we can sort of replicate that biological defect in a model system, using rats or mice or fish or even human neurons, and you can do things with these kinds of models that you obviously can't do in humans -- and I'll talk about some of those details.

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But then it helps you really understand what's -- what's going on in terms of the brain. Where actually are the defects occurring. What's the problem in terms of nerve cell communication. And then eventually you can start thinking on that basis what types of --
(Video playback ended.)
BY MR. WATTS:
Q. Now, back when you had a beard in 2017, is that what you said?
A. Oh, yeah. And I still say that.
Q. Good beard. I like it.
One more. Exhibit 458, November 16, 2017.
(Document marked for identification as Exhibit Kolevzon 458.)
(Video played.)
DR. KOLEVZON: And so the ways that you want to try to

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measure and sort of have your animal studies inform your clinical studies and then have your clinical studies inform your animal studies, that's what's called translational science, right.

(Video playback ended.)

BY MR. WATTS:

Q. Is that something you said back in 2017?

A. Yep.

Q. Let's go to 2020, October 7, 489.

(Document marked for identification as Exhibit Kolevzon 489.)

(Video played.)

DR. KOLEVZON: To develop new treatments, we've adopted the following approach.

First, we identify a specific gene that causes autism when mutated. Then we replicate

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the genetic defect in the model system using an animal or even a brain cell derived from a patient's blood cells.

We can use these models to better understand the biology and understand what's going wrong with brain cell connections. Then we study the various treatments, but, first, in the model, to see if they can reverse the biological changes associated with the genetic defects. If the treatments work in the models, then we move to clinical trials in humans affected by the same genetic changes.

There's enormous promise in this approach and many --

(Video playback ended.)

BY MR. WATTS:

Q. Is that part of what you said in 2020?

A. Yes. And I'd like to just

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provide some context here, right, because what we're talking about is translational science, where we use model systems that are extremely valuable in developing and testing new treatments, because we can do things in a model system and manipulate models in a way that we can't manipulate in humans. And it can provide us with evidence of proof of concept, right?

We want to think about, oh, this treatment might work in the model. Maybe it will work in humans. But in no way does the model recapitulate human condition. If, at best, it recapitulates some of the biology.

Q. And it's true you can use both human and animal studies to show that something is occurring, right?

A. I'd say, sorry, but broad and vague statement. Could you be more specific.

Q. I'll give you a specific example.

A. Thanks.

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Q. Let's go back to the Katz paper.

MR. WATTS: Exhibit 491. Pages 2 and 3.

BY MR. WATTS:

Q. "Human and animal studies have shown that maternal prenatal metabolic syndrome includes increased adiposity and insulin resistance and results in an inflammatory state, as well as altered leptin signaling. These changes have significant impact on fetal neurodevelopment secondary to neuroinflammation and can affect synaptic plasticity, oxidative stress, as well as neurotrophic and neuroprotective signaling."

Is that part of what you wrote in 2021?

A. I wrote that. And I think in this case it's important to see that the animal studies provide a window that's worth hypothesis testing, just like the animal studies with treatments

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provide a window.
But if you do a treatment study in a human being, no matter what the animal studies show, if the treatment fails in the human being, you wouldn't continue to say, oh, that treatment works because it worked in animals.
You'd only say it works if it worked in the humans. And you wouldn't be able to say it on the basis of one study. You'd have to say it on the basis of multiple studies across multiple sites.
Q. Doctor, let me show you Exhibit 445.
(Document marked for identification as Exhibit Kolevzon 445.)
BY MR. WATTS:
Q. This is a paper that you wrote with Theresa Tavassoli, titled, "Measuring Sensory Reactivity in Autism Spectrum Disorder: Application and Simplification of a

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Clinician-Administered Sensory Observation Scale."
Do you see that, sir?
A. Yes.
MS. BROWN: I just gave him the hardcopy, so take a minute to look if you need to.
BY MR. WATTS:
Q. And let's go to Page 288.
And my only point of bringing this up is on the first column, you all write, "In general, sensory questionnaires are considered valuable screening tools for sensory issues and have the advantage of being low cost and easy to administer," right?
A. Oh, yeah.
Q. And on the bottom of 288, second column, and 289, you write, "The Autism Spectrum Quotient, AQ, was used to screen autism spectrum disorder traits in the TD group. The AQ is a 50-item questionnaire with five subscales measuring autistic traits, social skills,

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attention switching, attention detail, imagination, and communication. Results from the AQ have been replicated cross-culturally and across different age groups with good test/retest reliability."
Did I read that correctly?
A. Yeah. The AQ is a good screening tool, but it's not a diagnostic measure.
Q. Now, in addition to that, you and I have already talked about the two most common parent report tools are the Modified Checklist for Autism in Toddlers, or the M-CHAT-R, right?
A. I don't know that we've talked about that, but --
Q. Do you agree?
A. -- that is a correct statement.
Q. And childhood autism -- Childhood Autism Spectrum Test, or CAST, is also used broadly, right?
A. I don't know that it's used

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broadly, but it's been used.
Q. And then we've talked about, before, ADOS, the Autism Diagnostic Observation Schedule, right?
A. I don't think we've talked about it before, but that is definitely a gold-standard diagnostic tool.
Q. And in your previous report in the Daniels-Feasel case, Exhibit 479, you said, "ADOS has improved our ability to detect."
Do you agree?
TRIAL TECH: What page?
THE WITNESS: Sorry. Can you finish the sentence.
MR. WATTS: Page 10 of 94. It's down at the bottom.
BY MR. WATTS:
Q. You write that, "The Autism Diagnostic Observation Schedule, ADOS, and the Autism Diagnostic Interview-Revised have improved our ability to test for autism, right?
A. I think that's true, yes.

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Q. Now, have you ever used the population-based cohort in Sweden called PAGES?
A. I have been part of collaborations that have used PAGES, yes.
Q. Okay. What other Swedish, Danish, European population-based cohorts have you worked in?
A. Numerous ones. I can't recall sitting here.
Q. Have you ever studied any of those cohorts with respect to acetaminophen?
A. So we may have queried some of those samples. I don't recall whether we have specifically.
Q. Okay. Doctor, we talked a little bit about the fact that third parties fund research that Mount Sinai does; is that right?
MS. BROWN: Objection to the form.
THE WITNESS: So I'll speak to my own research and the

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research of the Seaver Autism Center. It's funded by foundations. It's funded by the federal government. It's funded by industry.
BY MR. WATTS:
Q. Are you aware of a legislatively required database known as openpayments.com?
A. I am, yes.
Q. And if we could put up Exhibit 528 on the screen.
(Document marked for identification as Exhibit Kolevzon 528.)
BY MR. WATTS:
Q. Are you aware that since 2016, various Janssen industries have made payments to Mount Sinai Hospital exceeding $10 million?
MS. BROWN: Object to this document.
Did you make it?
MR. WATTS: I did.

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MS. BROWN: Okay. I object.
THE WITNESS: I am not aware nor do I follow the payments that are made to Mount Sinai Hospital. I follow the payments that are made to me.
BY MR. WATTS:
Q. So, as you understand the purpose of OpenPayments data, it's required by the federal government so people can know how much money a particular pharmaceutical company has spent to fund research at a particular hospital facility, right?
A. I understand that's the purpose of the law, yes.
Q. Okay. And, again, have you ever gone on there and seen how much money came from the Janssen entities to Mount Sinai?
A. I have --
MS. BROWN: Objection to form.
THE WITNESS: I have not,

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no.
BY MR. WATTS:
Q. Okay. Do you know, if we added 2023 to it, how much more money Janssen has paid Mount Sinai in payments?
MS. BROWN: Objection to form.
THE WITNESS: I do not know.
BY MR. WATTS:
Q. As we look at Exhibit 510.
(Document marked for identification as Exhibit Kolevzon 510.)
BY MR. WATTS:
Q. Mount Sinai has a data -- or a website that's entitled, "Our Partners, Our Relationships With Industry Provide Access to Partners and Funding Opportunities."
Can you see that?
A. Yes.
Q. And first up in terms of the list of our partners is Johnson & Johnson, right?

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A. That's on the list among many industry partners, yes.
Q. In addition to Johnson & Johnson, has Mount Sinai received gifts from Royalty Pharma?
MS. BROWN: Objection to the form.
THE WITNESS: I would have no way of knowing that.
MR. WATTS: Let me show you Exhibit 500, which is a Mount Sinai press release.
(Document marked for identification as Exhibit Kolevzon 500.)
MS. BROWN: Hang on. Let us get it, please.
BY MR. WATTS:
Q. You see it talks about a $20 million gift from Royalty Pharma?
MS. BROWN: Hey, I just want to give him the document before you start asking questions about it.

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MR. WATTS: Get 401 -- I mean, get 501, 502, and 503 while you are there.
(Document marked for identification as Exhibit Kolevzon 501.)
(Document marked for identification as Exhibit Kolevzon 502.)
(Document marked for identification as Exhibit Kolevzon 503.)
THE WITNESS: So this is an announcement about a gift that's meant to identify, interrogate, and combat health inequities by building a future that is more equitable for all communities, including those that are non-white, low-income, immigrant, uninsured and LGBTQ+.
BY MR. WATTS:
Q. And Royalty Pharma is a company, if you look at Page 4, that

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enjoys royalties on 35 commercial products, including Johnson & Johnson's Imbruvica and Tremfya, right?
A. That's what it says on the page.
Q. Let's go to Page 501 and look at the "Our History of Royal Pharma."
It says they "are the largest buyer of biopharmaceutical royalties and a leading funder of innovation across the biopharmaceutical industry."
And then it talks about "assembling a portfolio of royalties entitling us to payments on top-line sales of many of the industry's leading therapies, including Johnson & Johnson's Imbruvica and Tremfya."
Can you see that?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: I can see that it's written on the page.

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BY MR. WATTS:
Q. Doctor, as we go to 502 -- put that on the left and put 503 on the right.
MS. BROWN: Where did 502 come from?
MR. WATTS: I'll get you that. I --
MS. BROWN: And 503. Can we just identify what these are?
MR. WATTS: Yeah. If you go to the Google machine and turn it on, type in the top ten owners of Johnson & Johnson, this comes up.
MS. BROWN: Okay. So a lawyer did that and created these two exhibits?
MR. WATTS: No, I pulled it off the Google machine.
MS. BROWN: Okay. I object. These lack foundation.
MR. WATTS: I'll get you the URLs.
MS. BROWN: All right.

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BY MR. WATTS:
Q. On the left we see that the top four owners of Johnson & Johnson are the Vanguard Group, SSGA Fund Management, Inc., BlackRock Fund Advisors, and Geode Capital Management, all of whom are on the list of the top ten owners of Royalty Pharma.
Do you see that?
MS. BROWN: Objection. Lacks foundation.
THE WITNESS: I can say that I am totally unqualified and this is so outside of my scope, that I can read what's on the page, but I have no way of evaluating it.
BY MR. WATTS:
Q. Does Mount Sinai accept grants from third parties that are funded by pharmaceutical companies?
MS. BROWN: Objection to the form of the question.
THE WITNESS: I think that's a vague statement. And I can't

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say yes or no without more specifics.
BY MR. WATTS:
Q. I'm going to use an example. But I don't mean to suggest it's illegal, but you know what money laundering is?
MS. BROWN: I object to the form of the question.
THE WITNESS: I'm familiar with the --
BY MR. WATTS:
Q. Go ahead.
A. I'm familiar with the idea of money laundering.
Q. And that is routing the true source of the money through some third party so you can hide its true source, right?
MS. BROWN: I object to this entire line of questioning.
THE WITNESS: Again, I'm totally unqualified to be answering questions about money laundering.

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MS. BROWN: Also, who are we suggesting is money laundering?
MR. WATTS: I used it as an example.
BY MR. WATTS:
Q. Let me just ask you, are you aware of third-party entities through which Johnson & Johnson has routed payments to Mount Sinai on top of what we've already discussed?
MS. BROWN: I emphatically object to these questions as not based in facts or truth or relevance.
MR. WATTS: I know. We'll get to it.
THE WITNESS: I'm not aware now, no.
BY MR. WATTS:
Q. In terms of other pharma companies that pay you stipends, you are on the advisory boards for Ovid Therapeutics, right?
A. Yes.

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Q. RetroVirox Therapeutics?
A. I'm on the board. I do not receive a stipend.
Q. Do you have stock options?
A. No.
Q. Okay. Jaguar Therapeutics?
A. I'm an advisor to Jaguar Therapeutics, yes.
Q. Are you paid for your time?
A. I do get paid for my time if I bill for it.
Q. Okay. Are you paid for your time for RetroVirox Therapeutics?
A. I have not been paid for my time for RetroVirox.
Q. Do you expect to be paid?
A. I -- at some point in the future, if they are successful.
Q. Okay. You consult with Acadia?
A. I have consulted to Acadia in the past.
Q. Alkermes?
A. Alkermes, in the past, yes.

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Q. GW Pharmaceuticals?
A. I participated in an advisory board call for GW, yes.
Q. Were you compensated for your time --
A. Yes.
Q. Neuren Pharmaceuticals, or Neuren Pharmaceuticals?
A. I've consulted to Neuren.
Q. Clinibis Labs?
A. I have --
Q. I think I mispronounced that. Clinilabs --
A. Clinilabs, yes.
Q. -- Drug Development Corporation?
A. I have a consulting agreement. I haven't actually consulted with them yet.
Q. Okay. Scioto Biosciences?
A. I have consulted with them in the past.
Q. Seaside Therapeutics?
A. I've received grant support

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from Seaside, but I have not consulted with them.
Q. Okay. Hoffmann-La Roche?
A. I have received grant support for studies to do the project, but I haven't consulted with them, as far as I can remember.
MR. WATTS: If we can go back to 510, please. Second page.
BY MR. WATTS:
Q. Have you consulted with Merck?
A. Not that I recall.
Q. GSK?
A. I think GSK, or some subsidiary, funded studies that I was involved in.
Q. Okay. Is that Lan Bio on the right?
Do you know what that is?
A. I don't.
Q. Okay. Have you consulted with Eli Lilly?
A. I think Eli Lilly may have

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funded some studies that we did early on, but I don't consult to them, no.
Q. Novartis?
A. I don't consult to Novartis.
Q. Novo Nordisk?
A. I don't consult to Novo Nordisk.
Q. Roche?
A. I don't consult to Roche, as far as I know.
Q. Okay. But you do consult for Hoffmann-La Roche, right?
A. I think I have received funding for research, which is different than consulting.
Q. I can never tell the difference between the two, Hoffmann-La Roche, Roche.
Okay. Let me ask you this. How many litigations have you testified in, just ballpark?
A. I think total, between trial testimony and depositions, I think it's eight or nine.

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Q. Okay. Can you tell me what the McSweeney versus South Hampton Pediatric Associates is about?
MS. BROWN: Only if that's been disclosed.
MR. WATTS: It was. It was a prior testimony listed.
MS. BROWN: Okay.
THE WITNESS: I don't remember the details of that case. I remember that it was, broadly speaking, a medical malpractice case.
BY MR. WATTS:
Q. In Suffolk County, New York?
A. Correct.
Q. Do you know who the plaintiffs' lawyer who took your deposition was?
A. This was in New York, so I don't believe they have depositions, but I was in trial.
Q. I wish I lived in such a state.

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Tell me about the Ting versus Christina Ring case in Boulder, Colorado?

MS. BROWN: Same instruction.

MR. WATTS: Same answer. These are all prior testimonies.

BY MR. WATTS:

Q. Go ahead.

A. So I don't remember the details of the case other than to remember that it was a medical malpractice case.

Q. Anthem versus Franciscan Health -- Health System case in Washington?

A. I don't remember the details of that case at all.

Q. Okay. Hayes versus Johns Hopkins?

A. I don't remember the details.

Q. Anderson versus Johns Hopkins?

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A. Now, that one I should remember, because it was quite recent. That was a medical malpractice case, yes. I remember that case.

Q. Okay. What did it involve?

A. It involved a claim that a child who had hypoxic-ischemic encephalopathy due to some perinatal complication had autism.

Q. Okay. And when you said it was very recent, when were you deposed in that case?

A. I think that was in the beginning of the summer, maybe the end of the spring.

Q. And did they have the joy of deposing you in Kingston, New York?

A. No. They did it by Zoom, actually.

Q. Okay. Who took your deposition?

A. Oh, I remember the person's name. He had a -- can I get that as a multiple choice question?

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I can't remember the name. Sorry. He had an interesting name. Storm maybe.

Q. Sorry?

A. Storm maybe was his name. We can look it up.

Q. Maybe he's related to Stormy Daniels?

MS. BROWN: Things are going off the rails rapidly here. We are down to the bottom.

MR. WATTS: Segue there. Not me.

BY MR. WATTS:

Q. Okay. Let's go to Exhibit 506 and 544. Put them up left and right.

(Document marked for identification as Exhibit Kolevzon 506.)

(Document marked for identification as Exhibit Kolevzon 544.)

BY MR. WATTS:

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Q. Okay. Exhibit 506 is Bates page Kolevzon 1 through 108, and Exhibit 544 is Bates page Kolevzon 109 through 115.

And this is your so-called -- I'll just call it your correspondence file involving when you were talking to Steve Tillery?

A. Yep.

Q. And my first question is this:

What led to the second production in Exhibit 544 that wasn't included in the first that was Exhibit 506; do you know?

MS. BROWN: Objection to the form.

THE WITNESS: So sorry, can you -- I don't know the difference.

BY MR. WATTS:

Q. Well, the one on the left, 1 through 108, we got, and that was supposed to be your correspondence file

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involving -- involving Tillery?
A. Yeah.
Q. And then subsequently I got 544, which is Bates page 109 through 115. That's some more e-mails. And I'm just curious how that came down.
A. So I think the first time I was asked about this by the defense attorneys I only searched according to my inbox for Tillery.
Q. Yeah.
A. And the second time I was more comprehensive, so I included my sent items --
Q. Gotcha.
A. -- as well as my e-mails to Shanna Swan.
Q. Okay.
A. So there were some e-mails I think that were not included, based on my sent items.
Q. Now. Here is my question. If we look at the top of Exhibit 544, we see Shanna Swan's name, Alexander

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Kolevzon, Avi Reichenberg, David Kristensen, and Ann Bauer.
Do you see that?
A. Yes.
Q. Are those all individuals with whom you are familiar?
A. So I know Avi Reichenberg and I know Shanna Swan. I don't know David Kristensen other than paper, and I never heard of Ann Bauer before this case.
Q. Okay. Were you on calls with those individuals during the fall of 2022?
A. So I had one 30-minute call, and I'm certain that Shanna Swan was on the call and Stephen Tillery was on the call and Avi Reichenberg was on the call.
Q. Okay. And if we could, just put down 544 and go to -- go to 506. We'll go through it for just a second.
MR. WATTS: If you go back, to about 108. Let's go to 107. First bill. Yeah, right there.

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102.
MS. BROWN: What are these numbers? What is 102?
MR. WATTS: That's the Bates page number in the lower right-hand corner.
MS. BROWN: Of what exhibit?
TRIAL TECH: 506.
MR. WATTS: Of Exhibit 506.
MS. BROWN: Mine's not Bates'd.
BY MR. WATTS:
Q. Okay. Do you see on the screen that in Bates Number 102, we have the first entry on your billing records of being December 15th?
A. Yes.
Q. And that is a half an hour and a conversation with somebody at Butler Snow. Who was the individual at Butler Snow?
A. I believe it was David Snow -- sorry, David Cohen.
Q. Okay. And that wasn't the

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first time that you spoke with Mr. Cohen about this case, though?
A. No.
Q. How much earlier than December 15 was the first time that you spoke to Mr. Cohen?
A. I don't know exactly. It was within -- I don't know exactly. I don't want to guess.
Q. Well, was it in the month of November?
MS. BROWN: Objection to the form.
THE WITNESS: I think it was in December, but I don't know.
BY MR. WATTS:
Q. So with respect to that conversation --
A. Which conversation?
Q. The first conversation you had with David Cohen at Butler Snow, it was prior to December 15, 2022. We know that, right?
A. Yes.

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Q. Best estimate as to how many days prior to the first time where you actually billed your time?
A. I don't want to guess.
Q. Well, a week?
A. I don't want to guess.
Q. Okay.
A. I can -- if we look at the e-mail chain, I can sort of back-calculate it, but...
Q. Yeah. Okay. Go to Bates page number 100.
MS. BROWN: We need to find a way to match this up with what we have, because it's not matching up.
MR. WATTS: Okay. Bates page 100. At the top or at the bottom -- let's go to 101, and we'll come back to 100. You know how with e-mails you have to work backwards. So start with 101.
BY MR. WATTS:
Q. You see on November 30th,

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Tillery says to all five of you experts, "I'd appreciate having a call, WebEx, at your convenience, to bring you up-to-date and discuss next steps"?
A. Yes.
Q. That happens on November 30th?
A. Yes.
Q. If we go to the top of the screen, Ann Bauer responds, "I would be happy to participate"; is that right?
A. Yes.
Q. Do you see that?
A. Yes.
Q. Now, we go to 100, Shanna Swan says, "Me too," right?
A. Yep.
Q. And then on December 13th, Steve Tillery asks for a time for a call on WebEx, right?
A. Yes.
Q. And then on December 14th at 6:43 a.m., you say, "I'm going to bow out, folks. Good luck with the case,"

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right?
A. Right. But there's an e-mail in between where I also respond that I can't participate.
Q. Okay. And my question is, the e-mail in between -- and I want to say it was December 1, but we'll find it -- was that before or after you first talked to the Butler Snow lawyer?
MS. BROWN: Objection. Calls for speculation.
BY MR. WATTS:
Q. Go ahead.
A. So I believe it was before I spoke to the attorney, because I was involved in another case. And I remember speaking to those attorneys, and that was the impetus for backing out.
Q. Okay. The law firm defending the baby food case in Galveston was the one you talked to that was the impetus for backing out with Tillery?
A. Correct.
Q. Yes?

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A. Correct.
Q. Okay. And did you speak to David Cohen at Butler Snow before or after you talked to the law firm that was defending the Galveston case?
MS. BROWN: Asked and answered. Objection.
THE WITNESS: I think I spoke to David Cohen after I spoke to -- I think these two things happened separate from each other, and the conversation with David happened afterwards.
BY MR. WATTS:
Q. Okay. So as I get the sequence, you are talking with Tillery and Bauer and Shanna Swan?
A. Just to clarify, no. We had one 30-minute call, and then --
Q. I'm not going to take you through 100 pages of e-mails. But I -- I get it.
There was a discussion that happened where you answered 11 questions

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and --
A. No, I didn't answer 11 questions. There was a discussion for 30 minutes. I don't remember the details.
There were 11 questions on the agenda. I don't recall going through 11 questions. Then there was some back-and-forth in e-mails. An attempt to schedule a subsequent meeting, and then at that point I had backed out.
Q. And so at the time they were attempting to schedule a subsequent meeting, you have a conversation with the defense lawyer who is defending the Galveston autism case, right?
A. Correct.
Q. You identify that now maybe between the Galveston case and the acetaminophen case, you felt like you had a conflict?
A. Yes. It was a potential conflict.
Q. Okay. What was the nature

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of that conflict that you felt you had?
A. I think it was seen as problematic, potentially, to be on both defense and plaintiffs' side simultaneously in cases around exposures.
Q. Okay. I'm sorry, what?
A. In cases around exposures.
Q. Okay. So then after you backed out, following talking with the defense firm in the Galveston baby food case, you get a call out of the blue from Butler Snow asking you to consult with them in this case?
A. They didn't ask me to consult with them. They asked me if I would be interested in.
We had an introductory conversation. At that point I still hadn't looked at the literature.
Q. And at that point, after you spoke with Butler Snow, you sent the December 14th e-mail saying you're going to back -- bow out?
A. So, again, there was an

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e-mail prior to this, and I was still copied on these e-mails trying to schedule. So then I sent another e-mail to say, I'm going to have to bow out.
Q. Okay. And then the next day you had your first conversation where you billed Butler Snow for work on behalf of the acetaminophen manufacturers in this case, right?
A. Yes. I never saw myself as being retained by the plaintiffs' attorney. And at that point it was clear that I was going to be helping the defense attorneys.
Q. Now, let me take you to Exhibit 525.
(Document marked for identification as Exhibit Kolevzon 525.)
BY MR. WATTS:
Q. This is your billing file between 102 and 108. It's separate because -- it's part of your correspondence file, the other one, but I

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separated it.
A. Yeah.
Q. So here is my question. You had a 30-minute call on December 15th. A second 30-minute call on January 17th. Did no work in between, right?
A. Sorry, say that again.
Q. Yeah.
You had one call on December 15th for a half an hour, a second call on January 17th for a half an hour, and billed for no work in between, right?
A. Well, I billed for no work in between. Obviously, I did no work, but I billed for no work, so, yeah.
Q. That's my point.
Let's go to the next page.
The next thing that you do is on January 21st, you start preparing a report on changing prevalence rates.
Do you see that?
A. Yeah. Yes.
Q. And then on the 22nd, you

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start preparing a report on changing prevalence rates, correct?
A. Yes.
Q. So a total of four hours and 75 minutes.
A. Four hours and 45 minutes.
Q. I'm sorry, 4.75 hours, right?
Are you a football fan?
A. Do I have to answer?
Q. Yeah. Giants or Jets, which is it?
A. If I had to choose, I would go with the Giants.
Q. Okay. You know the Giants were playing on the 21st in the playoffs against the Eagles, right?
A. I can't confirm or deny.
Q. Did you watch them get killed by the Eagles 38 to 7 while you were working on your prevalence report?
A. I definitely was not watching the game.
Q. Okay. Well, you didn't miss

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anything, so there you go.
During the call on the 17th, did they ask you to write a report about changing prevalence rates?
A. During the call on the 17th of -- sorry. Can we go back?
Q. Call on the 17th, right there. Half an hour. You haven't done anything for a month. You have a call with Butler Snow for a half an hour and then you start working on a changing prevalence rate while the football games are on, right?
MS. BROWN: And I'm just going to object to the conversation on the 17th. It's been redacted for work product privilege. And so I'm going to instruct you not to answer what is underneath the redaction.
BY MR. WATTS:
Q. That's fair. That's fair.
The first thing that you chose to do, as it happens, right after a

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half an hour call with Butler Snow, is to write a report on changing prevalence rates, right?
A. So I was asked my opinions -- because that literature I had already reviewed. And I was asked my opinions on the changing prevalence rates, which I discussed, and then I was asked to provide some overview of those opinions in writing.
Q. And that's where you gave them the citation to Croen in 2002, right?
MS. BROWN: Objection to the form.
THE WITNESS: I don't know. And I don't remember when I first provided that citation.
BY MR. WATTS:
Q. Do you think if the football games hadn't been on, you might have spotted the 2003 Blaxill or 2003 Croen response?
MS. BROWN: Objection to the

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form.
THE WITNESS: I'm not sure how we can answer that question.
BY MR. WATTS:
Q. Let's see if we can just do one last thing and then we'll call this a day, okay?
A. Okay.
Q. If you would go to Document 404, which is your materials considered list.
(Whereupon, a discussion was held off the record.)
BY MR. WATTS:
Q. I'm going to hand you my copy of 404.
And what I need to know is, of the documents that you listed as your materials referred to, if you would take my blue pen and put a check next to each of them that you consider them to be a reliable authority.
MS. BROWN: I'm going to object to this as improper

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instruction for the witness. And it's an impossible task to be done in the remaining time.
BY MR. WATTS:
Q. I'll tell you what. Are there any authorities that you reference that you consider not to be reliable?
MS. BROWN: On materials referenced?
BY MR. WATTS:
Q. Sir?
A. I mean, I considered all these papers. In terms of the findings, I'd have to go through each and every one of them to determine whether they -- I consider them to be reliable --
Q. Right. That's what I meant.
A. -- on the basis of their design and methods and results.
Q. Which ones did you find to be reliable? And just put a check next to the ones that you found reliable.
MS. BROWN: Object to the form of the question.

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What part reliable?
BY MR. WATTS:
Q. Go ahead, sir. Just put a check on the ones that you think are reliable.
THE VIDEOGRAPHER: Just grab your microphone.
THE WITNESS: We'd have to go to the actual papers and make sure that I'm looking at all the papers, and then we can do that exercise.
BY MR. WATTS:
Q. Okay. Do you have any in here that you consider to be not reliable?
MS. BROWN: Same.
BY MR. WATTS:
Q. Just off the top -- top of your head?
MS. BROWN: Same objection.
THE WITNESS: You know, I don't want to respond off the top of my head. I'd rather look at

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the actual papers to make sure that I'm being thoughtful and comprehensive in my answer.
BY MR. WATTS:
Q. What's today's date, September 1st?
A. No -- oh, yeah. September 1st.
Q. Okay. I'll be asking you that question again. So if you want to think about that between now and the next time that we see each other, I think that would be a good thing for you to do. Okay?
A. Can you clarify the question.
Q. Sure.
MS. BROWN: There's no question. Don't worry about it. We'll look forward to seeing you.
BY MR. WATTS:
Q. There is very much a question that you're refusing to do, and that's okay.

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But the question is, I would like to know which of the materials you've considered, you consider to be reliable authorities.
A. I'm not refusing --
MS. BROWN: Okay. And to be fair -- hold on. Hold on.
He's not refusing. This is how you chose to spend your time today. He asked to be able to complete the task you've asked of him, to have each one of these studies so he can reliably go through them and answer your question.
MR. WATTS: Okay.
MS. BROWN: We don't have time for that.
MR. WATTS: Well, that's not my problem.
MS. BROWN: But I think it is, because it's your time.
MR. WATTS: Alli, you're not the judge. Now let me take my

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deposition, okay?
BY MR. WATTS:
Q. Now, my question is, which of these are reliable authorities. And if you'd like to answer it, fill it in before you sign your deposition, I'm happy to do that.
I'm making the request. I want a list of these materials considered that you consider to be reliable authorities, okay?
MS. BROWN: I object. He's not going to do that. He's already answered that question.
BY MR. WATTS:
Q. Go ahead.
A. So my answer is that I'm happy to do that.
Q. Okay.
A. However, I can't do that on the basis of titles. I need to do it on the basis of the actual articles. And there are, you know, 20 pages in here.
Q. Yep.

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A. So we'd have to go through each article, look at the methods, look at the rigor, look at the design, look at all the elements of it, and then I can try to answer that question.
Q. Okay. Great. Well, that's my request. And if you want to comply with it before you read and sign your deposition, you can. If you choose not to, whatever the repercussions are of that, that'll be fine.
MS. BROWN: There's no repercussions. I object to the instruction.
MR. WATTS: Judge, Judge, come on.
MS. BROWN: That's improper. Are you threatening him with repercussions? It sounded like a threat. It's outrageous.
MR. WATTS: It sounds like coaching.
THE WITNESS: I thought we were done.

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MR. WATTS: Objection to form. My goodness.
BY MR. WATTS:
Q. What's a business address and phone number that you can be reached at for purposes of identifying you on the record?
MS. BROWN: He's a retained expert. If you need to reach him, you call us.
THE WITNESS: I was going to say that.
MR. WATTS: Okay. So I'll list the business address of Skadden Arps?
MS. BROWN: Please do.
MR. WATTS: That's fine.
Okay. Those are all my questions. Thank you.
MS. BROWN: I'm going to have a few questions, but can we take ten minutes, please.
THE VIDEOGRAPHER: The time right now is 5:01 p.m. We are off

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the record.
(Short break.)
THE VIDEOGRAPHER: The time right now is 5:11 p.m. We're back on the record.
- - -
EXAMINATION
- - -
BY MS. BROWN:
Q. Welcome back, Dr. Kolevzon. How are you doing?
A. I'm good, thanks, Alli.
Q. Okay. Just a couple of quick questions for you at the end of the day here.
We started this morning talking about institutions and doctors you may know from different institutions. Do you remember those questions?
A. I do.
Q. And one of the things counsel was asking you about was things like, do you think Harvard is a good and respectable school, right?

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A. Right.
Q. And you agreed, of course, that Harvard and Yale and Mount Sinai, these are respectable institutions, correct?
A. Correct.
Q. And you were asked a number of questions about doctors that you may know and you may have written with or presented with or worked with over the years, correct?
A. Correct.
Q. And many of those doctors you believed to be good doctors, correct?
A. Correct.
Q. But, Dr. Kolevzon, I want to ask you about whether it is reasonable for doctors to have the opinion, based on the current scientific literature, that acetaminophen causes autism?
A. I don't think that the scientific literature supports an association between acetaminophen and autism, and it certainly doesn't support

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a causal one.
Q. And so is it possible, though, for reasonable scientists to disagree on the question of whether acetaminophen causes autism?
A. I think any reasonable scientist that would evaluate the literature would come to the same conclusion, so I think, in this particular case, it's not possible or it shouldn't be possible for a reasonable scientist to disagree.
Q. And you were shown, in connection with that line of questioning, you were shown a number of press releases and articles discussing whether or not there could be an association between acetaminophen and autism. Do you recall some of those?
A. Yes.
Q. Okay. And I want to show you and ask you some questions about Exhibit 560, which was this 2021 printout from a women's health section of the

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Harvard Medical School.
Do you recall some questions about this?
A. Vaguely, yes.
Q. Okay. And do you recall, actually, a lot of questions about just headlines and press releases and sentences and articles you weren't familiar with?
MR. WATTS: Objection. Leading.
THE WITNESS: I was asked to confirm that what was written on the page was in fact written on the page.
But, yes, they were from various press releases.
BY MS. BROWN:
Q. And is it important, though, when you're evaluating any kind of document, to actually read what the words in the document say?
A. Yes. I think in order to evaluate what the document is saying, you

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need to understand the source and the methods used to determine the conclusions.
Q. And so, for example, Exhibit 560's title, "Is a Common Pain Reliever Safe During Pregnancy?"
And let's just look briefly at what the article actually says.
Would you read for us, Doctor, the last two sentences of this press release that you were asked about the title of?
A. "While the issue they raise is important, it's worth noting that the concerns come from studies done in animals and human observational studies. These type of studies cannot prove that acetaminophen is the actual cause of any of these problems."
Q. And, Doctor, on the very next page of the Harvard press release, what does it say about the research on this topic?
A. It says the research on this

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topic is not conclusive.
Q. And is that consistent with your review of the literature?
A. It is.
Q. Okay. And what does it say in terms of whether or not this is an area where a causal association has been proven?
A. It says that more research is needed to confirm that this medicine is truly causing health problems, and to determine at what doses and at what points during the pregnancy exposure to acetaminophen might be most harmful.
Q. And was one of the sensible steps that the Harvard folks actually suggest pregnant moms do when considering whether or not to take acetaminophen during pregnancy?
A. It looks like they are recommending to consult with your doctor.
Q. Okay. And what is the concluding sentence, Doctor, of this Harvard press release as it relates to

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that advice to pregnant moms?
A. That assuming it's advised by your doctor, that the benefits outweigh the risks.
Q. Okay. You were asked some questions about the Mount Sinai website at Exhibit 520, and I just want to make sure that I understood your testimony and it's consistent.
Counsel asked you if you agreed with what Mount Sinai said on their website about acetaminophen.
Do you recall that question?
A. I do.
Q. Okay. Did Exhibit 520 that counsel showed you, titled, "Autism Spectrum Disorder," does it say anything about acetaminophen?
A. Not to my knowledge.
Q. Okay. And so he mentioned that this had been recently printed off of the Mount Sinai website. And there were some questions about whether you support what Mount Sinai says about

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acetaminophen.
But, in fact, this document doesn't say a word about acetaminophen, right?
A. Right.
Q. Okay. And what he did point you to is a section about causes, right?
A. Yes.
Q. The exact cause of autism isn't known; do you agree with that?
A. Other than genetics.
Q. Okay. Certain medicines taken during pregnancy may also lead to ASD in the child.
You talked to us about a study showing an association with valproic acid, correct?
A. Yes.
Q. And do you believe that there have been some studies showing a potential association?
A. Yes.
Q. Does this page of Mount Sinai's website say anything about

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acetaminophen causing autism?
A. Not that I'm aware of.
Q. Okay. You were asked a lot of questions, Dr. Kolevzon, throughout the day about a recent edition of a book chapter you wrote years ago that we have as Exhibit 494.
Do you recall that series of questions?
A. Yes.
Q. Okay. And when counsel was asking you about the questions, he referred to a -- what he termed to be a very recent edition of this book, correct?
A. Correct.
Q. And, in fact, this book, the addition that we have at 494, came out just within the last couple of years, correct?
A. Yes.
Q. Okay. I want to show you some -- and I understand you didn't write this section, right?

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A. No.
Q. Okay. Nevertheless, I want to show you some sections of this chapter, though, that counsel didn't show you and see if you agree with them.
First chapter of the paragraph says, "Caution against inappropriately causing the public to blame mothers for their child's condition is sometimes warranted in studies on associations in which a mother's agency is involved, such as the case of maternal antidepressant use during pregnancy."
Do you see that?
A. Yes.
Q. What does that mean to you, Doctor?
A. It means going back to the history of autism, people would blame mothers for their child's autism, and I think that's quite dangerous, especially as those theories have mostly been disproven.
And when a mother who is

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depressed takes an SSRI and passes on increased risk by virtue of their genetic susceptibility and tries to treat the depression and you blame the mother for taking the SSRI, that's potentially very damaging.
Q. This very recent chapter that you didn't write says on the next page, "Despite significant research into the association between conditions and complications of pregnancy and birth and autism spectrum disorder, the causal nature of these associations is still in question."
Do you agree with that?
A. Yes. I think that there are some associations that have been reliably shown, but the strength and consistency would not lead one to conclude that they are causal in nature.
Q. Okay. And you were pointed out this sentence on the next page about mechanisms, but I don't think the complete sentence was read.

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Would you read that final sentence for us there, Doctor.
A. "We present plausible biological mechanisms linking those risk factors to ASD and suggest some directions for future research."
Q. And what does "suggest future directions for future research" mean to you, Doctor?
A. It means that all these biological mechanisms that are proposed are hypothesis driven, and that's why it's important to do future research, to try to actually establish them as plausible.
Q. And is that consistent with your review of the literature, Doctor?
A. It is.
Q. Okay. And then I don't think we had a chance to actually look at what other authors said about acetaminophen. I want to ask you about that.
This short paragraph says,

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"It's also been suggested that acetaminophen increases the risk for autism spectrum disorder by causing neuronal oxidative stress. Only one meta-analysis has been published focusing on this association, and considering the susceptibility of individual observational studies to several biases, mostly confounding by indication, this association awaits further elucidation."
What does that mean, Doctor?
A. So, again, I didn't write this. But the person who did was able to review the literature at the time. Things have evolved since then, but I think they included one meta-analysis by Masarwa. And I think within that meta-analysis there were six or so studies included, only one of which used autism as an outcome.
And the kind of combined among all those studies odds ratio was something like 1.19. And so on that basis they are saying there is a possible

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association.
But because of the inconsistency across studies, a lot more work needs to be done to -- to establish that. And, actually, since then, multiple studies have examined this, and most have been negative.
Q. And anywhere in this short paragraph on acetaminophen, do these authors conclude that maternal use of acetaminophen is a cause of autism?
A. No, certainly not.
Q. Okay. And then, finally, Doctor, at the very end of this chapter, there is some conclusions and future directions.
Do you see that?
A. Yes.
Q. Okay. And I think we talked about some of these highlights. But let's just look at the very first unhighlighted sentence fragment.
"Although the etiology of autism remains largely unknown."

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Do you agree with that?
A. I think, other than genetics sort of en masse, it is largely unknown. And I think we still have many, many, many genes, even thousands, that we have yet to identify.
Q. Now I want to ask you a couple of follow-up questions, actually, about that.
We talked a little bit about genetics today, and I thought I heard you use the percentage 70 to 80 percent of autism may be caused by genetics.
Was that your testimony?
MR. WATTS: Objection. Form.
THE WITNESS: So we were talking about the heritability of autism, and I think, generally speaking, it's accepted to be about 80 percent, even perhaps more. I think in my report I said 70 to 90. So estimates range.
But that is the amount of

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the kind of clinical features or the phenotype that we see as being driven by genetics.
BY MS. BROWN:
Q. And how does that relate to the 20 percent or less of genes we've actually identified that you spoke about today?
A. Right. So 20 years ago we were able to identify 1 percent, 2 percent. Today we're able to identify 20 percent or even 30 percent.
What that means is that despite it being 80 to 90 percent genetic in origin, if you do a genetic test on a population of 100 kids, you're only going to find a specific gene in 20 to 30 percent. And that reflects the limitations in both the technology, our analytic methods, and just the state of the knowledge.
Q. And because we don't know what the genes are, does that mean that there's only a genetic cause or a genetic

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factor in 20 percent of those cases?
A. No. Decades and decades of literature, especially in twins, shows quite convincingly that 80 to 90 percent of autism is genetically driven.
Q. And, in fact, one of the things the folks who wrote this book chapter that we spent a lot of time on here today wrote was that "perhaps the most important potential confounder to consider is genetic susceptibility to ASD which may be associated with obstetrical suboptimality."
What does that mean?
A. So in this particular case, it means that the child may be at increased risk for autism based on in uterine conditions. Based on, you know, speculatively, stress of the mother. And it's for that reason that they are taking Tylenol or acetaminophen.
And it's not that the acetaminophen is causing the autism, it's the obstetric conditions that are causing

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the autism or some other genetic confounding.

There are studies that suggest that there are sort of genetic reasons why mothers might take Tylenol, and those same genetic reasons may drive the autism diagnosis.

Q. You were shown a Exhibit 545 today, which was a blank chart titled, "Kolevzon's Explanation for Rising Prevalence Rates of ASD."

Do you remember this chart?

A. Yes.

Q. Okay. And counsel started to fill in, in red, some dates.

A. Yes.

Q. Do you recall that?

A. Yes.

Q. Okay. And based on your 20-plus years of experience and your review of the scientific literature, are those dates reflective of the time period for which these explanations apply?

A. No. I think I mentioned

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that these explanations reflect dynamic changes over the last, in some cases, 40 years that have kind of iteratively affected prevalence rates.

So even though a certain mandate occurred in 2007, that doesn't mean that it only affected prevalence in 2007, because it affected prevalence from 2007 until today, for example.

Q. And so if you wanted to accurately put a date through which these explanations were continuing to influence rising prevalence rates of autism, what date would you put?

A. Today.

Q. Today for all of these, correct?

A. Well, every year that the CDC has monitored rates, it's gone up and up and up. So if the explanation, as I've proposed it or as it's been proposed by many, many, many people, and commonly accepted in the scientific community, you would see these -- the continued impact

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up until today.

It's the last prevalence rate that was estimated by the CDC is 1 out of 36 children has autism. And if that's because of some, as of yet, unknown environmental factor, it would be absolutely shocking.

Q. And, in fact -- and we'll mark this as Defense-1 and provide it to your deposition.

(Document marked for identification as Kolevzon Defense Exhibit 1.)

BY MS. BROWN:

Q. In fact, along those lines, if we just take a quick peek back at 520, that Mount Sinai website that counsel showed you. Mount Sinai says, "The increase in children with ASD may be due to better diagnosis and newer definitions of ASD. Autism spectrum disorder now includes syndromes that used to be regarded as separate disorders."

Is that part of what you

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were talking about regarding the prevalence rates, Doctor?

A. Yes. And Mount Sinai doesn't say that. I would say it's the consensus in the scientific community that these are the factors that have led, for the most part, to the increase in prevalence.

Q. But as it relates to these factors, you say the consensus in the scientific community, you were shown bits and pieces of a bunch of other articles talking about DSM-V and whether or not that influenced prevalence rates in the way you describe it. Help us understand that.

A. So in science you have conflicting findings. But the totality of the literature, the totality of the evidence and certainly the consensus among scientists is that this is mainly artifactual.

Q. A few quick questions on the e-mails that counsel was asking you about

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at the very end of your deposition.
Is it accurate that you were actually approached by the plaintiffs in this litigation at one point?
MR. WATTS: Objection. Form.
THE WITNESS: Correct.
BY MS. BROWN:
Q. Okay. And did you understand -- did you tell the lawyers representing the plaintiffs in this litigation that you believed acetaminophen could cause autism?
A. No. I told them that I was willing to investigate the literature, to help answer that question.
Q. Did you tell the lawyers representing the plaintiffs in this litigation that you ever formed the opinion that supports their litigation theory that maternal use of acetaminophen can cause autism?
A. No. In fact, as I recall, I told them that my previous investigation

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of another exposure determined that, in fact, the exposure was not causal.
Q. Were you ever retained by the lawyers representing the plaintiffs in this litigation?
A. No. As I recall, an e-mail was sent saying that they would send a retention letter. I never received a retention letter, and I didn't assume to be retained by them.
Q. Is your opinion based on who is hiring you?
A. Absolutely not. My opinion is based on my evaluation of literature, which is based on my experience scientifically and clinically.
Q. And similar to that you were asked a lot of questions about funding of scientific research and scientific articles.
Are the results or the efforts of your scientific endeavors determined by who is funding them?
A. No. I think it's always

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important to reveal and be transparent about potential conflicts of interest, which is what we do. Obviously, everybody comes to science with some biases that they have to be aware of.
But by no means is my science dictated, determined, or influenced by the funder. Or at least not the outcomes. Maybe the nature of the experiment can depend on certain priorities, but the outcomes are dependent on the data.
MS. BROWN: Dr. Kolevzon, that's all I have for you. Thanks so much for your time.
MR. WATTS: A little bit more.
- - -
EXAMINATION
- - -
BY MR. WATTS:
Q. Ready.
Dr. Kolevzon, counsel for the acetaminophen manufacturers asked you

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about my questions concerning respectable institutions, Harvard, Yale, Johns Hopkins; you recall that, right?
A. Yes.
Q. Then you said that you don't think that any reasonable science could disagree with you, right?
A. No. What I said is in the case of, specifically, acetaminophen causing autism, looking at the literature as it stands, I don't think a reasonable scientist could conclude that it causes autism.
Q. Now, she pointed you to 520, which was a Mount Sinai press release that didn't mention acetaminophen, but she didn't mention Exhibit 466.
MR. WATTS: Put that up.
(Document marked for identification as Exhibit Kolevzon 466.)
BY MR. WATTS:
Q. This is a Mount Sinai press release that does mention acetaminophen,

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right?
MS. BROWN: Objection. Misstates the document.
THE WITNESS: This is --
BY MR. WATTS:
Q. Is it a Mount Sinai press release?
MS. BROWN: Same objection.
BY MR. WATTS:
Q. Yes?
A. Yes. We'd have to look at the study, which is not --
Q. Is it titled, "Acetaminophen Use"?
The first word of the whole article is the first word of the title that mentions acetaminophen, right?
A. So this is a different web page, sponsored by a different site and has nothing to do with autism.
Q. This document has nothing to do with autism?
A. The study that the document is based on is language delay in girls.

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Q. So with respect to acetaminophen, 466 mentions acetaminophen, and it has a quote from Shanna Swan of the Icahn School, right?
MR. WATTS: Second page.
THE WITNESS: Okay.
BY MR. WATTS:
Q. And Carl-Gustaf Bornehag, a professor at Karlstad University and adjunct professor at the Icahn School.
MR. WATTS: Take that down.
BY MR. WATTS:
Q. There's Shanna Swan, and then right below that is Carl-Gustaf Bornehag, right?
A. So I'm familiar with the study.
Q. Okay.
A. This is a study that has nothing to do with autism.
Q. So Shanna Swan, Mount Sinai, says, "Pregnant women should limit their use of this analgesic during pregnancy." And the adjunct professor at Icahn says,

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"New data suggests the use of acetaminophen poses a risk for pregnant women," right?
A. I don't think that's a reasonable conclusion to draw based on the evidence.
Q. But it's a conclusion by reasonable scientists, right?
MS. BROWN: Objection. Misstates testimony.
THE WITNESS: I think if a reasonable scientist in this case evaluated the totality of the literature, they would not draw that conclusion.
BY MR. WATTS:
Q. And if we look at the consensus statement in 561, it was written in part by an Icahn scientist, Shanna Swan, right?
MS. BROWN: Objection to the form.
BY MR. WATTS:
Q. Yes?

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A. Yes.
Q. And the 91 physicians that were listed include Veerle Bergink, also an Icahn scientist in 562, right?
A. If Shanna Swan or Veerle Bergink evaluated the literature as it relates to autism, prenatal use, and acetaminophen, I don't think they could reasonably conclude that it causes autism.
Q. You know, that's why I used Exhibit 562. I showed you all the stuff that was evaluated by the consensus statement group.
Remember all those tables, five tables? Do you remember that?
A. Vaguely.
Q. I didn't take you through that for my health. That was all the stuff that they reviewed and listed what they reviewed before coming up with a conclusion that there was a need for precautionary action regarding paracetamol use during pregnancy, right?

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MS. BROWN: Objection. Argumentive.
THE WITNESS: Disagree with that conclusion.
BY MR. WATTS:
Q. Now, the difference between you and them is after your school came out with this press release, with this consensus statement, those folks looked at it and they weren't retained in litigation, right?
MS. BROWN: Objection to the form of the question as false.
THE WITNESS: I don't know if they were retained or not. They may have been retained for the plaintiffs.
BY MR. WATTS:
Q. Well, between the time that this consensus statement came out in 2018 until almost five years later when you decided to go to work for Butler Snow, did you do one lick of work with respect to acetaminophen, one lick other than

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have your name on a book chapter that you say you didn't write?
MS. BROWN: Highly argumentive. I object.
BY MR. WATTS:
Q. Go ahead.
A. I think I've made it clear that I did not start investigating the relationship between acetaminophen and autism, I did not find it plausible, until recently.
Q. Did you ever tell Mr. Tillery that you didn't find his theory plausible? I didn't see an e-mail that said that.
A. I think in the first e-mail that I sent to Shanna Swan, I reflected some skepticism but also agreed, nevertheless, to dig into the literature, because I was curious about the science.
Q. Now, since the Butler Snow people have called you, your bills total $82,250 as of a month ago. Do you know how much time you spent this month?

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A. I don't know how much time.
Q. What is your best estimate?
A. I don't want to guess.
Q. Well, I mean, you've been here eight hours. How much time did you prepare this week?
A. Several hours a day.
Q. Yeah. You remember the supplemental thing. You've read all the deposition reports -- I mean all the expert reports, right?
A. Yes.
Q. And then you read all the rough transcripts of the depositions, right?
A. I spent a lot of time, yes.
Q. What's a lot of time? What's your best estimate?
A. I don't want to make a best estimate.
Q. Okay. Would you be willing to send me your supplemental bill for this time so we can have an accurate description of what you billed as of the

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date of the deposition?
A. Sure --
MS. BROWN: Counsel.
THE WITNESS: I'm sure defense counsel will provide it to you.
BY MR. WATTS:
Q. Now, let me ask you this next thing, this caution against blaming mothers. Did you hear one word out of my mouth saying I blame the mother?
A. No.
Q. Now, if the mother is not warned about a risk, can she make an informed choice as to whether to take a pharmaceutical product or not?
MS. BROWN: I object to the hypothetical.
THE WITNESS: I'm not in the position of advising mothers whether to take medicine or not.
BY MR. WATTS:
Q. The label that mom gets when she goes to Walgreens or CVS or Walmart

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doesn't say one word about autism, does it?

MS. BROWN: Objection to the form of the question.

THE WITNESS: I'm a child psychiatrist. I don't prescribe medicine to --

MS. BROWN: Let him finish, please. Let him finish.

BY MR. WATTS:

Q. Yeah, but you saw it doesn't say anything about acetaminophen causing autism, does it?

MS. BROWN: Objection to form. Lacks foundation.

THE WITNESS: To my knowledge, the label of acetaminophen does not say it causes autism. Rightfully so.

BY MR. WATTS:

Q. Okay. Do you know what the label over in Europe says about the relationship between acetaminophen use and autism spectrum disorder?

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MS. BROWN: Objection to the form.

THE WITNESS: I'm not a regulatory expert. I don't prescribe medicine to pregnant women, and I don't practice in Europe.

BY MR. WATTS:

Q. Now, if a label says that you should use this as judiciously as possible given this risk, do you think moms are just going to ignore that label?

A. Again, I'm not in a position to judge moms. I'm not in a position to evaluate labels.

Q. You would agree that there is absolutely no basis to blame mom for taking acetaminophen when she's not told one word about acetaminophen risk of autism, agreed?

MS. BROWN: I object to this whole line of questioning as lacking foundation.

And, Counsel, you are out of

Page 628

time.

BY MR. WATTS:

Q. Go ahead.

A. Again, I don't find myself in the position of advising moms.

Q. Okay. Well, you made the comments about moms, so...

MS. BROWN: Counsel, I'll give you the courtesy of a couple more questions, but you're over seven hours.

MR. WATTS: Well, I have the same -- no, it's seven hours the first part, then I get the same time as you. That's what the order said, but I'm not --

MS. BROWN: Okay. Okay.

BY MR. WATTS:

Q. Let me just ask you, with respect to whether something is causal or not, does cigarette smoking cause cancer?

MS. BROWN: Objection to the form.

THE WITNESS: I'm not here

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to provide testimony on the relationship between cigarette smoking and cancer.

BY MR. WATTS:

Q. But assuming you were asked in front of the trial, do you have an opinion as to whether it causes cancer, yes or no?

MS. BROWN: Asked and answered.

THE WITNESS: I don't feel comfortable providing an expert opinion on that.

BY MR. WATTS:

Q. Okay. Fair enough.

With respect to 545.

MR. WATTS: Put it on the screen.

By MR. WATTS:

Q. And counsel wrote in something about today and all that stuff, and we'll have that.

I'm just curious. We've been here for eight hours. Are you able

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to tell me about the percentage of the increased prevalence that is due to any of the five factors that you talked about?

MS. BROWN: You asked that nine hours ago. I object.

THE WITNESS: So I am not able, as I sit here, to isolate these five factors and tell you what percentage of a rate increase it is.

But we are able to look at the rate increase and we are able to see that the majority of that increase is due to these methodological factors.

BY MR. WATTS:

Q. Oh, and 12 percent of it is because of younger age of diagnosis. What other diagnosis rate increase are you going to give to any one of these so that we can add it up and see if it's close to 100 percent or not?

MS. BROWN: Asked and

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answered like nine times now.

THE WITNESS: Yeah, I think I've answered that question. I am not able, at this moment in time, to mathematically model the rate of increase per item.

BY MR. WATTS:

Q. Now, next-to-last issue. She asked you whether the plaintiffs' lawyers in this litigation approached you. You understand that Mr. Tillery has nothing to do with this litigation.

A. I have no understanding whatsoever of Mr. Tillery's role in this litigation or not.

Q. You are testifying in an MDL proceeding in the Southern District of New York; you know that, right?

A. Yes.

Q. And you know that Mr. Tillery is not one of the co-leads, is not on the plaintiffs' executive committee, not on the Plaintiffs' Steering Committee, has nothing to do

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with this litigation?

MS. BROWN: I object to the form as lacking foundation.

THE WITNESS: The only -- no, I don't know what his role is --

BY MR. WATTS:

Q. Do you know whether --

MS. BROWN: Wait, wait, let him finish, please.

BY MR. WATTS:

Q. Do you know whether Mr. Tillery has even filed a single lawsuit?

A. I have no idea what Mr. Tillery's role is.

Q. Okay. So when she says the plaintiffs' lawyers in this litigation, in order to be in this litigation, you have to file a single lawsuit, right?

MS. BROWN: Objection to the form.

THE WITNESS: I have a sense that you're arguing with --

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BY MR. WATTS:

Q. Nope.

A. -- Ms. Brown.

Q. I'm not.

A. So, I'm sorry, what's the question?

Q. So this idea that plaintiffs' lawyers in this litigation approached you. Did I approach you?

A. No.

Q. Did Ashley Keller approach you?

A. No.

Q. Did Mark Lanier approach you?

A. No.

Q. Did any members of the plaintiffs' executive committee and the MDL appointed by the court approach you?

MS. BROWN: Objection. Lacks foundation.

THE WITNESS: No. I have no idea.

BY MR. WATTS:

Q. Did any members of the Plaintiffs' Steering Committee appointed by the court approach you?

MS. BROWN: Same objection.

THE WITNESS: I have no idea.

BY MR. WATTS:

Q. Okay. Now, this question about your funding sources, do the results of your scientific endeavors relate to who funded them? You've done no scientific research on the relationship between acetaminophen and autism outside of the litigation context in this case; is that true?

MS. BROWN: Asked and answered.

THE WITNESS: I have not done research on the relationship between acetaminophen and autism outside of the context of this.

BY MR. WATTS:

Q. And in your CV, updated however well it has been, it has grant

applications with respect to grants that are ongoing right now, right?

A. Yes.

Q. Have you sought any funding from any third party to do scientific research with respect to the relationship between acetaminophen and autism?

A. I have not myself, no.

MR. WATTS: Okay. That's all my questions. Thank you.

MS. BROWN: Nothing further. Thank you so much, Doctor.

THE VIDEOGRAPHER: The time right now is 5:42 p.m. We are off the record.

****

(Excused.)

(Deposition concluded at approximately 5:42 p.m.)

CERTIFICATE

I HEREBY CERTIFY that the witness was duly sworn by me and that the deposition is a true record of the testimony given by the witness.

It was requested before completion of the deposition that the witness, ALEX KOLEVZON, M.D., have the opportunity to read and sign the deposition transcript.

_____

MICHELLE L. GRAY,
A Registered Professional Reporter, Certified Court Reporter, Certified Realtime Reporter and Notary Public
Dated: September 5, 2023

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INSTRUCTIONS TO WITNESS

DATE: September 5, 2023

Please read your deposition over carefully and make any necessary corrections. You should state the reason in the appropriate space on the errata sheet for any corrections that are made.

After doing so, please sign the errata sheet and date it.

You are signing same subject to the changes you have noted on the errata sheet, which will be attached to your deposition.

It is imperative that you return the original errata sheet to the deposing attorney within thirty (30) days of receipt of the deposition transcript by you. If you fail to do so, the deposition transcript may be deemed to be accurate and may be used in court.

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ACKNOWLEDGMENT OF DEPONENT

I,_____, do hereby certify that I have read the foregoing pages, 1 - 640, and that the same is a correct transcription of the answers given by me to the questions therein propounded, except for the corrections or changes in form or substance, if any, noted in the attached Errata Sheet.

_____
ALEX KOLEVZON, M.D. DATE

Subscribed and sworn to before me this
_____ day of _____, 20____.
My commission expires:_____

_____
Notary Public

LAWYER'S NOTES

PAGE LINE

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WORD INDEX